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VOL. VIII. (NEW SERIES) NOS. 1 & 2 JANUARY AND APRIL, 1945

JOURNAL OF NEUROLOGY, NEUROSURGERY AND PSYCHIATRY

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ISSUED BY THE BRITISH MEDICAL ASSOCIATION

LONDON: BRITISH MEDICAL ASSOCIATION HOUSE, TAVISTOCK SQUARE, W.C.1

Published Quarterly. Subscription 25/- per annum. U.S.A. $6.00 Post Free.

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Brown, A. B. (1845). *Rev. Neurol.*, 168, 427.

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THE JOURNAL OF
NEUROLOGY, NEUROSURGERY AND PSYCHIATRY
FOUNDED BY S. A. K. WILSON

VOL. 8. [New Series] JANUARY–APRIL, 1945 Nos. 1 and 2

METABOLIC FACTORS AFFECTING FIBRILLATION IN DENERVATED MUSCLE

BY

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(From the Department of Anatomy, University of Oxford)

(RECEIVED 11TH JANUARY, 1945)

Introduction

FIBRILLATION, the spontaneous rhythmical twitching in voluntary muscles following denervation, has been the subject of many observations since Schiff (1851) first described the phenomenon. Some workers have dealt with the time of onset and distribution of the activity, and others have concerned themselves with the unit in action and the frequency of the contractions. The electrical activity accompanying the twitches has also been investigated and a number of observers have been concerned with the chemical factors which influence fibrillation. Finally the relation of fibrillation activity to muscle atrophy following denervation has received considerable attention.

In this paper we present the results of a study, by means of action potential records, of the relation between muscle fibrillation and metabolic activity. Different species of experimental animal have been used and observations have also been made from experimentally denervated muscles in man.

A Review of the Literature

(*a*) *The Direct Observations of Denervated Muscle.*—Schiff (1851) was the first to give an adequate description of the phenomenon of fibrillation. His observations were made on the tongues of dogs after section of both hypoglossal nerves. He notes that for three days after nerve section the tongue is completely paralysed. Between three and five days after section, a few spontaneous surface twitchings are seen. These rapidly increase in number until the whole tongue is in a state of continuous agitation. He says that the tongue as a whole appears to be undulating but that this is due to summation of the spontaneous asynchronous rhythmic twitches of individual muscle bundles. After three or four months the activity is somewhat weaker and at the end of six months has ceased; the tongue is then pale and grossly atrophied. In the rabbit, fibrillation commences between the third and fourth day, earlier than in the dog, and Schiff relates this to the loss of " electrical conductivity " of the severed nerve fibres which also, he says, disappears earlier in the rabbit. He also makes the observation that fibrillation occurs in limb muscles after interruption of their nerve supply, but that it can only be seen when the skin and fascia are removed. He says of the limbs, that air or other external stimulus increases fibrillation activity, but that this is not a constant phenomenon. Finally, he notes that after section of the facial nerve in the rabbit the vibrissæ of the affected side start to quiver on about the fourth day. When the nerve regenerates, this spontaneous quivering ceases.

Following Schiff's observations, the phenomenon was recorded again by both Rogowicz (1885) and Ricker (1901), but they added nothing to the account given by Schiff. Langley and Kato (1914–15*b*) re-investigated the phenomenon in some detail. They confirmed Schiff's general observations, and added the following details. The onset of fibrillation is established by the fifth day in the limb musculature of the rabbit and is best seen by reflected light from the muscle surface. As a rule, fibrillation at any one spot has a constant rhythm. The rate, rhythm, and intensity of contraction vary considerably in different parts of the surface of the same muscles, but the phenomenon is generally similar in both red and white muscles. Fibrillation ceases a few minutes after death, before the phenomenon of indirect excitability disappears. The rapidity of fibrillation is lessened by feeble circulation, and the surface application of adrenalin for this reason may stop it. Indeed, if the blood-flow through a muscle be stopped for ten to fifteen minutes, on restoration of circulation fibrillation remains greatly diminished or absent.

In a later paper Langley (1917) mentions that fibrillation is increased by warmth and decreased by cold, but gives no details of experimental findings in this connection. He also states that the activity

varies from muscle to muscle; for instance, in the cat it is always less in the soleus than in the gastrocnemius, although he did not find this particular difference in the rabbit. He observes that a few fibrillary twitches can be seen in the cat for a considerable time after death and he believes that he and Kato "did not observe for long enough on the rabbit." He also finds that fibrillation is slight in muscles in the later stages of recovery following denervation.

Langley and Kato (1914–15*a*) and Langley and Hashimoto (1918) observe that fibrillation varies in different parts of the same muscle, but that it is relatively constant at any one spot. The contracting unit they believe to be very localized, 0·5 to 1 mm. in length, and from this they infer that a portion only of a muscle fibre is involved in the contraction. Eccles and his collaborators (1941) have shown conclusively, however, that the whole of the muscle fibre is involved in the contraction.

Hines and Knowlton (1933) state that, following division of the sciatic nerve in the rat, typical fibrillary contractions are seen as early as the third day after denervation in the gastrocnemius and continue for at least forty-two days, which was the longest period during which observations were made. Denny-Brown and Pennybacker (1938) confirm previous observations that fibrillation commences in the cat's gastrocnemius on the fifth day at the earliest.

Tower (1939) has reviewed the literature on the subject and states that she has observed fibrillation in a muscle kept experimentally denervated for a year. She has also shown (1941) that, after isolation of the lumbar spinal cord and dorsal root section in the monkey, fibrillation does not occur within two weeks of the operation in the paralysed muscles unless, in addition, the sciatic nerve or other lower motor neuron is sectioned. Solandt and Magladery (1942) made similar observations after high thoracic cord section with dorsal root section in rats, but it is interesting to note that on the side opposite to that, on which the sciatic nerve was cut, transient muscle atrophy and increased acetylcholine sensitivity were recorded. Solandt, Partridge and Hunter (1943) noticed that following the fixation of a skeletal muscle (nerve supply intact), atrophy took place, and although there was an increased sensitivity to acetylcholine, fibrillation did not occur. The fixed muscles contracted only occasionally, and after the second day were usually flaccid. They suggest that, if fibrillation is due to an increased sensitivity to acetylcholine, as claimed by Denny Brown and Pennybacker, these fixed muscles should have exhibitied the phenomenon. Reid (1941) has shown that denervated muscles in the frog do, on occasions, fibrillate, and has made the interesting observation that the phenomenon never occurs before the forty-sixth day and, moreover, is only seen in "summer" frogs, i.e. those operated on between November and January (Australia). He also notes that fibrillation in one frog was increased from 100 to 140 twitches per minute on the application of warm Ringer solution.

(*b*) *The Electrical Activity accompanying Fibrillation of Denervation.*—Schäffer and Licht (1926) noted fine oscillations of the string galvanometer when recording from four-days' denervated tongue musculature in the dog, but their apparatus was relatively insensitive and thus the form of the action potentials was uncertain.

Proebster (1928) described spontaneous action currents recurring at intervals of about 1/9 second, which he obtained from the relaxed weak left biceps muscle of a thirteen-year-old boy who had probably suffered a traumatic birth injury to the brachial plexus. Recordings from normal muscles of the opposite side of the body showed no such action potentials. He also reports that action potentials of a similar type can be obtained from cases of long-standing post-poliomyelitic paralysis. Proebster considered that these action potentials were due to periodic discharge from the central nervous system. It is, however, more probable that they were fibrillation action potentials.

Brown (1937) has described the electrical activity of denervated (cat gastrocnemius) muscle recorded with concentric needle electrodes. It consists of a continuous series of diphasic action potentials repeating at from two to seven times per second. The insertion of the needle excites a number of fibres, and for a while rapid discharges of some 30 per second may be encountered. The highest voltage recorded was 30 microvolts, and Brown remarks how close this figure is to that recorded from single muscle fibres excited by acetylcholine (25 microvolts). This, he says, leads to the conclusion that these spontaneous action potentials are to be attributed to the activity of single muscle fibres.

Rosenblueth and Luco (1937), also using cats, recorded from tongue, face, and leg muscles five to forty days after denervation. They also used concentric needle electrodes. They found that from all the denervated muscles they studied a continuous asynchronous series of spike potentials could be led off with concentric needle electrodes. The spontaneous activity was greatest 10 to 30 days after nerve section. They also describe the effect of drugs on the spontaneous activity (*vide infra*). Denny-Brown and Pennybacker (1938) have summarized the literature relating to the action potentials from denervated muscles. They have shown that action potentials in no way differing from those obtained from completely denervated muscles in animals can be obtained from the affected muscles of patients suffering from amyotrophic lateral sclerosis, or from facial palsy with partial recovery. Tower (1939) also summarizes the literature on the action potentials from fibrillating muscle, and finds that the rate of contraction of the unit responsible for the activity is around 9 per second in the cat. This rate remained unchanged throughout a year of observation; only the amplitude of the action potential decreased as the muscle atrophied.

(*c*) *The Effect of Drugs on the Activity of Fibrillation of Denervation.*—Langley and Kato (1914–15*b*)

state that curare and nicotine are without effect on fibrillation in the gastrocnemius of a rabbit. Physostigmine does not cause any marked increase in spontaneous fibrillation, and curare after physostigmine does not cause any marked decrease. They were not certain that these drugs had no effect. Langley (1915–16) states that calcium chloride causes the cessation of fibrillation when applied locally or parenterally, and Langley (1917) found that the injection of pituitary extract, although rendering the muscle pale, was without effect on fibrillation, whereas the surface application of adrenalin decreased the activity markedly.

Frank, Nothmann and Hirsch-Kauffmann (1922) have shown that the intravenous injection of acetylcholine increases greatly the spontaneous fibrillation of the denervated dog's and cat's tongue. Brown (1937) has shown the increase of fibrillation activity in response to the intra-arterial injection of small doses of acetylcholine; large doses give rise to "electrical silence."

Rosenblueth and Luco (1937), using cats, confirm Brown's findings, and, in addition, show that atropine has no effect on spontaneous fibrillation activity; both eserine and prostigmine cause a transitory increase in activity. In addition, both eserine and prostigmine, especially the latter, sensitize muscle to acetylcholine. They also state that in the early stages following denervation (5–6 days), acetylcholine even in large doses only increases spontaneous fibrillation activity and no contracture takes place. Fifteen to 30 days after denervation large doses of acetylcholine cause contracture and "electrical silence." It thus appears that the contracture response to acetylcholine develops more slowly than the contraction response. Finally they found that relatively small doses of curare were required to reduce markedly the effects of the intravenous injection of acetylcholine.

Denny-Brown and Pennybacker (1938) showed that a drop of saline containing acetylcholine, diluted 1 : 500,000, allowed to fall on the surface of a muscle just commencing to fibrillate on the 7th day after nerve section, brought about an immediate local contraction lasting some twelve to twenty seconds. As the contraction subsided, all the muscle fibres in the area remained fibrillating for many minutes, and gradually the disturbance passed by *imperceptible transition* of moderate fibrillation into the original state of a few isolated fibres fibrillating.

Solandt and Magladery (1940) tested the influence of a large number of drugs, anæsthetics, and procedures such as electrical exercise, adrenalectomy, etc., on the spontaneous fibrillation activity of the rat's gastrocnemius-soleus group. They recorded the fibrillation action potentials with needle electrodes through the skin, in addition to checking their results by visual examination of the muscle on a number of occasions. They found electrical recording to be more sensitive than visual inspection. Anæsthetics such as ether and the barbiturates were without effect on fibrillation activity, as were atropine, curare, and eserine. Potassium chloride, in doses which are frequently lethal, temporarily abolishes fibrillation; calcium chloride temporarily arrests the activity but there is rapid adaptation. Quinine hydrobromide abolishes the activity for one-half to four hours whereas quinidine sulphate is effective for nine hours. Acetylcholine given intravenously in comparatively large doses can abolish the activity for 10 to 30 minutes.

Levine *et al* (1942), also using the denervated gastrocnemius-soleus complex of rats, obtained results somewhat different from those of Solandt and Magladery. They ordinarily used the method of visual inspection of the muscles although in some experiments action potentials were recorded. In particular they found that atropine, in addition to quinine and quinidine, inhibited fibrillation. They also found that ether and nembutal anæsthesia inhibited the activity 30 minutes and longer following induction. They found that prostigmine noticeably increased the activity.

Magladery and Solandt (1942) have shown that spontaneous fibrillation is increased by the intra-arterial injection of small concentrations of potassium chloride. The excitation, like that produced by acetylcholine, is abolished by quinidine in doses which arrest spontaneous fibrillation. They suggest that acetylcholine, potassium, or both may be the casual agents of fibrillation of denervation.

(d) The Relation of Denervation Atrophy to Fibrillation Activity.—Langley (1915–16) was of the opinion that fibrillation contributes markedly to denervation atrophy, even if it is not the only factor involved. In support of this is the higher oxygen consumption of denervated rabbit's muscle compared with normal muscle (Langley and Itagaki, 1917). Knowlton and Hines (1934), however, failed to find a raised oxygen consumption in denervated rat's muscle up to 28 days. Langley and Hashimoto (1918) say of rabbits that the degree of fibrillation seen at the time of death corresponds in general to the degree of atrophy of the muscle, but there are exceptions; for instance, the fibrillation in the soleus is usually less than might be expected in relation to its atrophy. This confirms previous observations in the cat (Langley, 1917). Solandt and Magladery (1940) prevented fibrillation in the rat's denervated gastrocnemius-soleus complex with quinidine, but found that the loss of weight due to atrophy was still marked. They concluded that fibrillation was not responsible for the denervation atrophy. Levine *et al.* (1942) on the other hand, state that the administration of prostigmine, which increased spontaneous fibrillation in the denervated gastrocnemius-soleus complex of the rat, was accompanied by an increase in the rate of atrophy by 47 per cent.; by contrast, atropine, which decreased spontaneous fibrillation, was accompanied by a decrease in the rate of atrophy by 39 per cent. Fischer (1939), however, has shown that electrically induced exercise decreases muscle atrophy following denervation and also increases the oxygen consumption of the affected muscle.

Summarizing this review of the literature, there is general agreement as to the main features of fibrillation activity, but there are some aspects which are not yet satisfactorily explained. For instance, the time of onset of fibrillation varies in different species of animal. In general, the onset is more rapidly established in small animals, but no analysis of the factors responsible for the variation has been made. In addition, the fact that fibrillation activity differs from muscle to muscle in the same animal and even in different parts of the same muscle has not been accounted for, but it is clear that workers using the method of visual inspection have been more concerned with this variation than those using the method of electrical recording through the intact skin.

The literature contains contradictory statements on the effect of drugs, particularly with regard to atropine. However, in no case have observers reported that the factor of local or body temperature has been taken into account during the course of their experiments. Discrepant findings have also been reported with regard to the relation of denervation atrophy to fibrillation, although the weight of evidence is against any direct relationship between the two phenomena.

Material and Methods

Material.—The laboratory animals used were mice, rats, guinea-pigs, rabbits, and an adult rhesus monkey. In addition, one of us (R. E. P.) submitted to a series of observations carried out on the experimentally denervated brachio-radialis and extensor carpiradialis muscles in his left forearm.

Operative Procedures.—1. In the mice, rats, guinea-pigs and rabbits, the sciatic nerve was exposed in the thigh under intravenous nembutal anæsthesia, the sural nerve was separated from the sciatic complex, and the tibial and peroneal nerves were crushed 10 times with smooth-bladed forceps at the mid-femur level. In two rabbits the peroneal nerve only was crushed at its point of entry into the anterior tibial muscle group, and in one rabbit 3 cm. of the peroneal nerve were resected and the muscle group which it supplies was kept denervated for two years.

2. In the monkey, the right radial nerve was exposed in its course in the radial groove of the humerus under ether anæsthesia. The nerve was crushed 10 times with smooth-bladed forceps.

3. In one of us (R. E. P.) under local anæsthesia, the nerves of the left brachio-radialis and extensor carpiradialis longus muscles were crushed 10 times with smooth-bladed forceps 5 cm. from the points of entry into their respective muscles.

Recording Apparatus.—The action potentials are led off with concentric needle electrodes (Adrian and Bronk, 1929) in all cases. The insulated core of the needle is connected to the amplifier through a lead shielded by earthed flexible steel gas piping, itself in electrical contact with the hypodermic needle. The amplifier and recording apparatus follow conventional lines. An all-mains high and low tension supply is used. Hum is eliminated by feeding high tension to the pre-amplifier valves through a four-stage resistance capacity smoothing circuit; the filaments being supplied with smoothed rectified alternating current. Resistance capacity networks are also necessary to eliminate disturbances due to 1–5 cycle modulation of the mains voltage. The pre-amplifier feeds voltage and power amplifiers driving respectively a cathode ray oscilloscope and loud speaker. The frequency response of the amplifier is linear from 30 to 2,000 cycles. There is a small falling off between 2,000 and 4,000 cycles. The low-frequency cut-off makes it possible to record action potentials within less than half a second of the insertion of the needle electrode into the muscle.

Recording Technique.—With the amplifier switched on, the needle electrode was pushed rapidly through the skin into the denervated muscles. Recordings were made from the day following denervation up to functional recovery. In animals submitted to peroneal and tibial nerve crushing, recordings were made from the anterior tibial group of muscles; in the monkey, they were made from the extensor communis digitorum muscle, and in man, from both the muscles which had been denervated. A number of recordings were made from different parts of the selected muscles on each occasion. Anæsthetics were not used for making recordings, for the procedure is less painful than giving a hypodermic injection. Functional recovery was estimated in the usual way in man and monkey. In the remaining animals the toe-spreading reflex was found to be the most reliable guide, although it cannot be directly related in time to the onset of functional recovery in the anterior tibial group (Altschul and Turner, 1942). Care was taken that the room temperature in which the recordings were made was between 18° C. and 22° C. and the animals were brought into the room three hours before observations were made.

Experimental Observations

(1) The Electrical Activity of Normal Muscle

It is necessary to give a brief description of the electrical activity recorded by concentric needle electrodes from normal muscle in order to contrast it with that obtained from denervated muscle.

The insertion of the electrode into a normal muscle in any of the animals used—including man—causes a brief outburst of electrical activity which in no way differs from that due to motor unit activity related to reflex or voluntary movement. The number and frequency of the action potentials and duration of the outburst depend both on the speed and depth of insertion of the electrode, and we have assumed that it is caused by the mechanical stimulation of terminal nerve fibres. It is heard as a rumble from the loud speaker. When the electrode is at rest in the muscle and the animal quiet, there is "electrical silence." When muscles contract, the motor unit activity gives rise to action potential spikes up to 1 millivolt in amplitude and from 5 to 10 milliseconds in duration (Fig. 1); these are also heard as a series of low-pitched sounds. The description given of motor unit activity in normal muscle in man by Weddell *et al.* (1944) is in general applicable to animals. Mono- di-,, and triphasic spikes and occasional polyphasic spikes, which are of high amplitude, are seen. The amplitude of motor unit action potentials in the same muscle varies, but lies between 100 microvolts and 1 millivolt; the duration is from 5 to 10 milliseconds. The only notable difference is the smaller average amplitude and shorter average duration of the action potentials obtained from corresponding muscles in the smaller animals (Fig. 2). This leads to a series of rather higher pitched sounds being heard from the loud-speaker in small as compared with large animals. In small animals, fewer muscle fibres must compose motor units in corresponding muscles, for we have found, on histological examina-

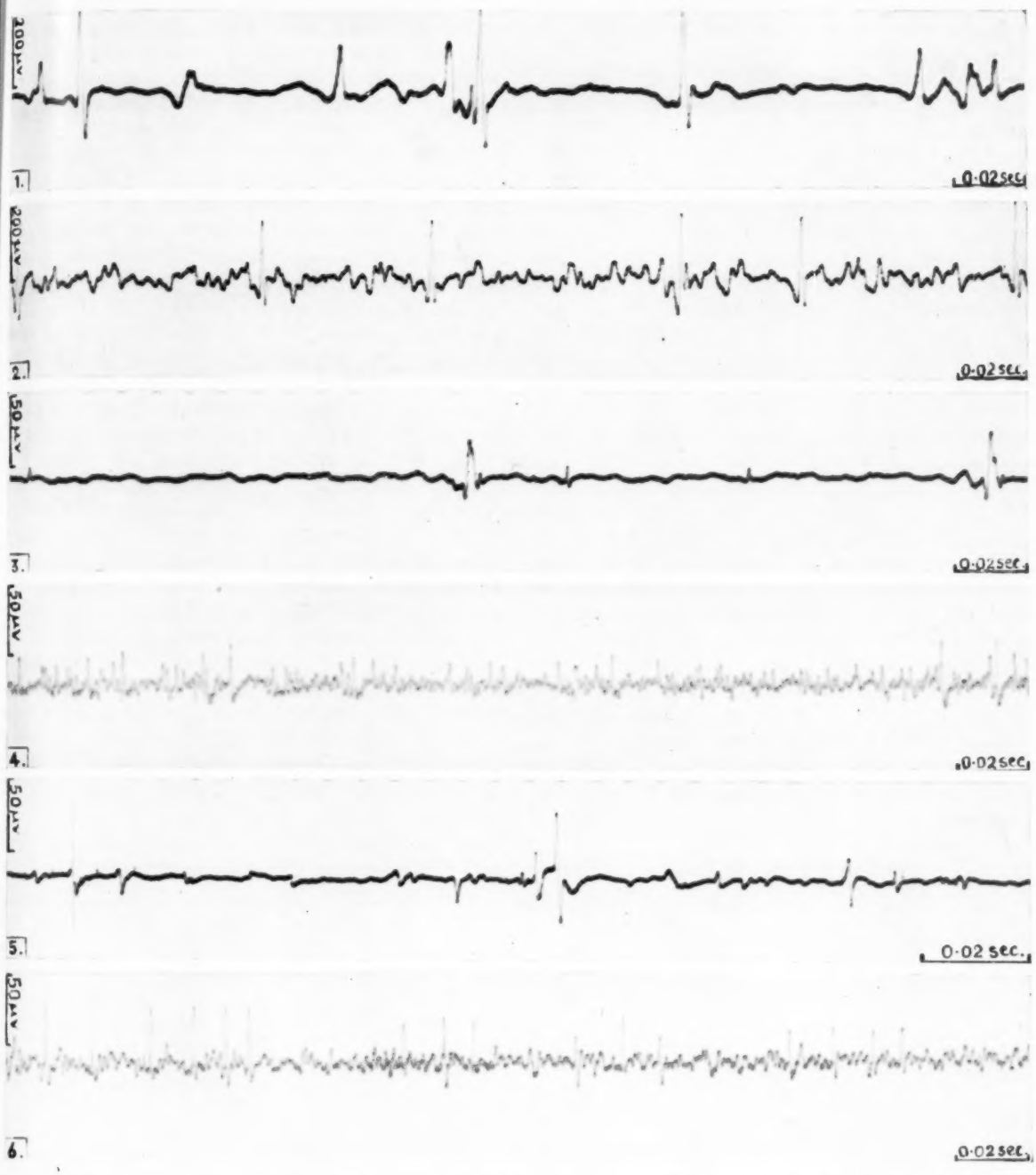

FIG. 1.—Motor unit action potentials from a normal human brachio-radialis muscle during a minimal voluntary contraction.
FIG. 2.—Motor unit action potentials from the normal tibialis anterior muscle of a guinea-pig. The muscle was only contracting feebly.
FIG. 3.—A motor unit and fibrillation action potentials from an 8 days denervated tibialis anterior muscle (rabbit).
FIG. 4.—Fibrillation action potentials from the tibialis anterior muscle of a mouse denervated 6 days previously.
FIG. 5.—Fibrillation action potentials from a human brachio-radialis muscle denervated 30 days previously.
FIG. 6.—Fibrillation action potentials from the tibialis anterior muscle of a rabbit weighing 1·5 kilogrammes 20 days following denervation.

tion, that individual muscle fibres vary but little in diameter in the species of mammal referred to in this paper. We have related this fact to our findings in man, that in muscles which have to perform delicate movements (i.e. face and larynx) motor units are composed of fewer fibres than in the limb muscles, and give rise to correspondingly smaller and sharper action potentials (Weddell *et al.*, 1944).

(2) The Time-course of the Electrical Activity in Denervated Muscle

1. *The Activity aroused by insertion of the Needle Electrode.*—Following denervation, no spontaneous motor unit activity is recorded from muscles, but the insertion of the electrode still gives rise to an outburst of motor unit action potentials. This mechanically aroused activity can be provoked for

varying times in the different species of animal used; for instance, in the mouse it can be obtained for 48 hours and in man for 18 days (see Table I). The larger the animal the longer this activity can be aroused. As already stated, it has been assumed that the activity in the intact muscle is due to stimulation of terminal nerve fibres by the needle point, but this can hardly be true in man two weeks after denervation. It may be that the motor unit is a morphological entity which retains its identity for a considerable time after denervation, and responds as a whole to mechanical stimulation. This mechanically aroused motor unit activity rapidly decreases in amount with the onset of fibrillation. Fibrillation action potentials appear at varying times after denervation, depending upon the size of the animal under observation; in small animals (mouse) they appear within 48 hours, in man they do not appear for 16 days (see Table I).

At first fibrillation action potentials only appear on insertion of the needle electrode and overlap by varying periods the mechanically aroused motor unit action potentials. They take the form of mono- or diphasic spikes 3 to 100 microvolts in amplitude and 1 to 2 milliseconds in duration and are heard as sharp clicks from the loud-speaker. It is difficult to distinguish both visually and audibly the chief type of action potential early in the overlap period, particularly in the mouse. Within 12 hours of the commencement of change of type of action potential, however, the two varieties can be clearly distinguished (Fig. 3). When motor unit action potentials disappear, spontaneous fibrillation action potentials make their appearance. A few days after the appearance of spontaneous fibrillation action potentials, insertion of the electrode evokes a large number of high frequency fibrillation action potentials (frequencies up to 40/second have been recorded) which gradually die away in the course of 3–4 seconds, leaving a residue of 4–5 spontaneous repetitive fibrillation action potentials.

(2) *The Spontaneous Activity.*—This increases in amount (number and frequency of the action potentials) for two weeks following its commencement, but the recording methods used do not allow of a quantitative estimate of the increase. It is also significant that the amount of activity is related to the size of the animal under investigation. For instance, it is far more vigorous in the mouse than in man (see Figs, 4 and 5). Moreover, in rabbits of the same breed, the amount of fibrillation obtained is smaller in large animals of the same age (see Figs. 6 and 7). Spontaneous fibrillation activity has been obtained in the anterior tibial group of muscles of a rabbit which had been kept denervated for two years. The average number of action potentials recorded is, however, somewhat less than in recently denervated muscle (Fig. 8). The frequency of spontaneous fibrillation action potentials when fully developed is from 2 to 10/second. In man, atrophy was easily recognizable clinically within two weeks of operation, and the circumference of the forearm had diminished by 2 cm. but spontaneous fibrillation had not yet commenced.

TABLE 1.—THE TIME-COURSE OF THE ELECTRICAL ACTIVITY OF DENERVATED MUSCLES

Species and number of animals	Weight in kg.	Motor unit action potentials: mechanical	Fibrillation action potentials: mechanical	Fibrillation action potentials: spontaneous	Motor unit action potentials: voluntary or reflex	Functional recovery	
						Days after operation	Days after appearance of first motor unit action potential
Mice (5)	40–45 gm.	Up to 3 days. Precise time difficult to estimate.	20 hours	40 hours	8 days	11	3
Mice (5)	45–50 gm.	Up to 3 days. ditto	24 hours	48 hours	9 days	12	3
Guinea-pigs (5)	300–450 gm.	Up to 3 days. ditto.	40 hours	2 days	26	30	4
Guinea-pigs (5)	450–600 gm.	Up to 3 days. ditto.	48 hours	3 days	29	34	4
Rabbits (10)	1·5–2·0 kilos.	Up to 8 days. ditto.	4 days	5 days	38 days	45	7
Rabbits * (10)	2–2·8 kilos.	Up to 8 days. ditto.	5 days	7 days	41 days	53	12
Rabbits (2)	2·0 and 2·1 kilos.	Up to 8 days. ditto.	4 days	5 days	13 days	22	9
Monkey (1)	6·8 kilos.	Up to 8 days. ditto.	8 days	9 days	42 days	49	7
Man (1)	76·3 kilos.	Up to 18 days. ditto.	16 days	18 days	55 days	57 and 70	2 and 15

* Nerve crushed close to muscle.

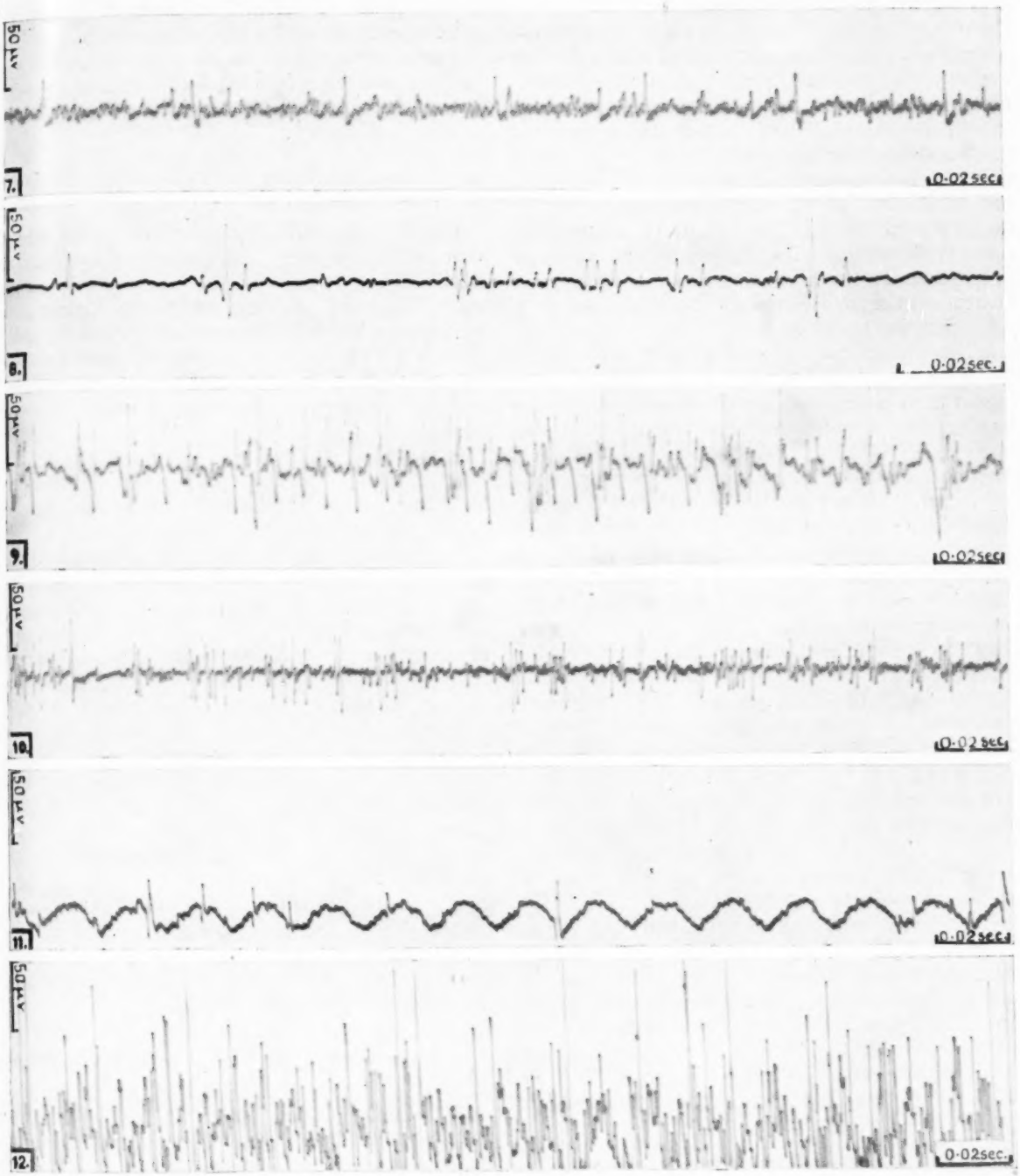

FIG. 7.—Fibrillation action potentials from the tibialis anterior muscle of a rabbit weighing 2·8 kilogrammes 20 days following denervation

FIG 8.—Fibrillation action potentials from the tibialis anterior muscle of a rabbit 2 years following denervation.

FIG. 9.—Fibrillation action potentials from the tibialis anterior muscle of a thyroidectomized rabbit 20 days following denervation.

FIG. 10.—Fibrillation action potentials from the tibialis anterior muscle of a rabbit fed on desiccated thyroid 20 days following denervation.

FIG. 11.—Fibrillation action potentials from the tibialis anterior muscle of an anæsthetized (ether) eserinized rabbit 14 days following denervation. (When this record was made the needle electrode had been in position for one half-hour.)

FIG. 12.—As for Fig. 11, but three seconds after the injection of 20 mg. of acetylcholine intravenously. The position of the electrode was unaltered.

In cases where the nerves are crushed, spontaneous fibrillation continues unabated until about two weeks before motor unit action potentials first appear, when a marked decrease in the number of the action potentials recorded occurs. It has not been possible to measure this decrease quantitatively for it would require an excessive number of needlings and recording material, but it is very definite. The

first motor unit action potentials appear close to the point of entry of the nerves into the muscles, and subsequently spread from these points through the muscles. Between 5 and 6 days after the first motor unit action potentials are recorded in the rabbit, the activity has spread throughout the muscle group. An appreciably longer time (12 days) was recorded in man, and shorter time (3 days) in the mouse due to the size of the muscles under examination. During the spread of motor unit activity, fibrillation virtually disappears, although in some cases a few fibrillation action potentials can be recorded from isolated areas for as long as a year following re-innervation.

When motor unit action potentials first appear following re-innervation, they are not sustained for longer than a few seconds at a time; moreover, they are of low amplitude and short duration and can only be distinguished from fibrillation action potentials in their relation to voluntary or reflex movement. Some weeks following re-innervation, motor unit action potentials assume their characteristic form, although their range of amplitude is greater than in normal muscles and a considerable number of polyphasic action potentials of medium amplitude are seen (Weddell *et al.*, 1944). Functional recovery in the animals submitted to sciatic crush cannot be detected until some time after the appearance of motor unit activity. We have found that the toe-spreading reflex appears about the same time as the anterior tibial muscle group can be felt to contract.

In the monkey the early stages of functional recovery were difficult to assess, so that the figures given in Table I are only approximate. In man, in the brachio-radialis muscle, the first onset of functional recovery was stated by the subject to have occurred on the day the first motor unit action potentials were elicited. Two days later minimal movements were observed. The assessment of recovery was, however, easier in the case of this particular muscle owing to its having become adherent to the overlying scar. In the extensor carpi radialis muscle, which was not attached to skin, recovery was not obvious for some days. A more detailed analysis of our findings in man has already been given (Weddell *et al.*, 1944).

In the rabbits in which the peroneal nerves were crushed close to their entry into the muscle groups, there was no essential difference in the time-course of electrical activity.

(3) Factors Influencing the Time-course of Fibrillation of Denervation

(1) *The Effect of Thyroidectomy.*—Our finding that the time of onset and quantity of spontaneous fibrillation in denervated muscles depends on the size of the animal and not the species, suggests that a metabolic factor is concerned in the activity. We therefore submitted 5 rabbits to thyroidectomy 4½ weeks before the tibial and peroneal nerves were crushed. This period was chosen as the result of the observations of Marine (1922) that the metabolic rate following thyroidectomy in rabbits is by this time lowered 35 to 40 per cent. Our observations are given in Table II, and show clearly that thyroidectomy delays the onset of fibrillation activity.

A curious finding which we are at a loss to explain is that the amplitude and frequency of fibrillation action potentials after thyroidectomy, both spontaneous and mechanically aroused, are greater than in normal control animals. The frequency of the action potentials is sometimes high enough to give rise to musical notes from the loud-speaker (Fig. 9).

(2) *The Effect of Feeding Desiccated Thyroid.*—Four grams of desiccated thyroid were fed daily to two rabbits of known weight for a week, by which time they had started to lose weight daily. The peroneal and tibial nerves were then crushed in the usual way. On the day following denervation, the insertion of the electrode gave rise to a more vigorous outburst of motor unit activity than in the control animal, but the same was true of muscles with intact nerve supplies. Fibrillation commenced earlier in the animals fed with thyroid and was more vigorous when fully established than in control animals, although not so vigorous as that in thyroidectomized animals (Fig. 10). Table III summarizes our findings.

(3) *The Effect of Temperature.*—In our pre-

TABLE II.—THE TIME-COURSE OF THE ELECTRICAL ACTIVITY OF DENERVATED MUSCLES.

(Thyroidectomized rabbits)

Species and number of animals	Weight in kg.	Motor unit action potentials: mechanical	Fibrillation action potentials: mechanical	Fibrillation action potentials: spontaneous	Motor unit action potentials: voluntary or reflex	Functional recovery	
						Days after operation	Days after appearance of first motor unit action potential
Rabbits control (4) ..	1·6–2	up to 7 days	4 days	5 days	38 days	45	7
Rabbits thyroidectomized (5)	1·9–2	up to 8 days	8–10 days	12–14 days	30 days	34	4

TABLE III.—THE TIME-COURSE OF THE ELECTRICAL ACTIVITY OF DENERVATED MUSCLES

(Rabbits fed on desiccated thyroid)

Species and number of animals	Weight in kg.	Motor unit action potentials: mechanical	Fibrillation action potentials: mechanical	Fibrillation action potentials: spontaneous	Motor unit action potentials: voluntary or reflex	Functional recovery	
						Days after operation	Days after appearance of first motor unit action potential
Rabbits control (4) ..	1·6–2·0	up to 7 days	4 days	5 days	38 days	45	7
Rabbits fed on dessicated thyroid (2) ..	1·62–1·7	up to 3 days	2 days	3 days	36 days	43	6

liminary experiments with rabbits we compared the electrical activity with the activity seen visually on each occasion. In this way we confirmed the observations of previous workers and were able to establish conclusively that the visual picture was reflected in the action potential picture. Following these preliminary observations we conducted a second series of experiments with the skin intact. Our observations under these conditions were far more consistent and, although we found that fibrillation did vary from place to place in a muscle, the variation was far less than when the muscle was exposed; moreover, the difference in the average activity in various muscles was not so marked as that reported by Langley (1917) using the method of observation of the exposed muscle.

In the next series of experiments we tried the effect of warming and cooling exposed fibrillating muscles, both locally and as a whole. We found that the activity was extremely sensitive to temperature change. Bathing with cool Ringer's solution (18° C.) stopped the activity, which was restored by bathing with a solution at 38° C. Exposure to the air in a cool room (15° C.) for five minutes stopped the activity, but it was restored on shining a lamp (100 watt bulb in an "Anglepoise" lamp 2 ft. distant) on the muscle for one minute. It rapidly died away again on removal of the lamp. Further experiments showed that cold air currents would stop a local region of muscle fibrillating. A reduction of 1·5° to 2° C. in local muscle temperature (measured by buried thermo-couples) was effective in the rabbit. The effect of warmth and cold was next tried without removal of the skin, by applying warm (39° C.) and cold pads (18° C.) around the denervated limb of a rabbit. The electrode was first inserted in the muscle and a steady stream of impulses recorded; on application of the cold pad, the activity gradually died away and within five minutes had ceased. It gradually returned as the limb grew warm again, but was rapidly restored by the application of the warm pad. The assessment of the quantity of fibrillation activity by inspection is clearly liable to grave errors unless precautions are taken to keep the muscle moist and at a *uniform* temperature.

The Effect of Drugs on Fibrillation of Denervation

We have re-investigated the effect of a few drugs under controlled temperature conditions in the rat. In particular we were interested in the effect of atropine, for opinion in the literature is divided as to its effects. Table IV summarizes our results. We find that our observations coincide with those of Solandt and Magladery (1940) but are at variance with those of Levine *et al.* (1942). In addition we tried the effect of prostigmine intravenously in an unanæsthetized rabbit. 2·5 mg. were injected into an ear vein at the same time as recordings were being made from the left anterior tibial group of muscles denervated 2 weeks previously. There was a rapid increase in fibrillation activity which reached a maximum in 120 seconds, and lasted for some 15 minutes. The rabbit became weak following the injection and could not maintain its upright position, and the limbs felt quite flaccid. All normal muscles were fasciculating vigorously at this time. Recovery was complete within 35 minutes of the injection.

TABLE IV

Drugs	Effect of fibrillation determined visually and by action potential records	Dose (rats, 150–210 gm.)
Ether anæsthesia.	Nil.	In full anæsthetic doses.
Nembutal anæsthesia.	Nil.	In full anæsthetic doses.
Atropine sulphate.	Nil.	Up to 100 mg.
Quinine.	Abolishes the activity up to 5 hours.	20 mg.
Quinidine sulphate.	Abolishes the activity up to 10 hours.	25 mg.

Finally in 3 rabbits (weighing approximately 2 kilogrammes each) with sciatic nerves crushed two weeks previously, recordings were made from the denervated muscles with the animal under nembutal anæsthesia and at constant temperature. 20μg of acetylcholine were injected intravenously.

There was no measurable increase in fibrillation. 0·2 mg. of eserine were next injected, again without obvious effect. The acetylcholine injection was then repeated. There was a great increase of fibrillation activity which reached a maximum within 3 seconds (Figs. 11 and 12). In 30 seconds the activity had returned to normal. These experiments confirm the observations of Rosenblueth and Luco (1937).

Discussion

Summarizing our observations we have shown that the time of onset of fibrillation following denervation varies with the size of the mammal; the smaller the mammal the earlier the onset. We have shown that the quantity of fibrillation decreases as the size of the animal increases. Thyroidectomy delays the time of onset of fibrillation, while the administration of desiccated thyroid advances it. Warmth increases fibrillation; cold decreases it. In short, we have shown that fibrillation is dependent on a metabolic factor. Finally, we have demonstrated that from the clinical point of view denervation atrophy is detectable before fibrillation action potentials are recorded. These observations probably account for some of the inexplicable and discrepant findings in the literature reviewed above. They certainly explain the varying time of onset of fibrillation in different species of mammal.

The effect of temperature on fibrillation activity is presumably directly related to metabolism, and we have shown that very small changes have a profound effect on the activity. This probably explains the puzzling variations in the activity reported by Langley and his collaborators. It may also account for the decreased activity resulting from the surface application of adrenalin, an effect not shared by pituitrin given intravenously, although both drugs cause visible vaso-constriction (Langley and Kato, 1914–15*b*). It probably also explains why fibrillation activity was found to last so much longer after death in the dog than in the rabbit (Langley, 1917).

Reid's observations are of particular interest in relation to our findings. It had long been thought that fibrillation does not occur in poikilothermic animals, but it is now clear that in "summer" frogs fibrillation does occur 46 days following denervation. The long interval between denervation and the onset of fibrillation has previously been related to species differences, and not to a metabolic factor.

The discrepancies in the literature relating to the effect of drugs, particularly atropine, may have been due to a neglect of the temperature factor during the observations, for we have found using both electrical and visual methods of recording that atropine has no effect on fibrillation in the rat. It will be remembered that Solandt and Magladery used chiefly the visual method. In addition, the observations of Levine *et al.* on the effect of anæsthetics are probably related to a lowered temperature or metabolic rate, for decreased fibrillation activity was not detectable until approximately 30 minutes after the induction of anæsthesia.

Finally, we have confirmed the findings of Solandt and Magladery that there is no close relationship between atrophy and fibrillation, for muscle atrophy in man can be observed before the onset of fibrillation.

It is clear that the method of action potential recording with concentric needle electrodes is a valuable means of detecting the presence of denervated muscle fibres. It can thus be used to determine minimal lesions of motor nerves. It can also be used to demonstrate minimal degrees of reinnervation following motor nerve interruption and subsequent regeneration. In each case the determination can be made without exposure of the muscle concerned. The clinical application of action potential recording with concentric needle electrodes has already been given (Weddell *et al.*, 1944).

Summary

(1) The time-course of fibrillation of denervation is dependent on the size of the mammal under investigation. The onset occurs in the mouse in 24 hours and in man (limb musculature) 16 days.

(2) Thyroidectomy in rabbits delays the onset of fibrillation following denervation.

(3) Feeding desiccated thyroid to rabbits decreases the time before the onset of fibrillation following denervation.

(4) The amount of fibrillation of denervation (number of frequency of action potential spikes) is greater in small than in large mammals, regardless of species.

(5) The amount of fibrillation depends on the temperature of the denervated muscle under investigation. A small fall in temperature causes a marked decrease of activity.

(6) Our observations on the effect of drugs on fibrillation confirm the work of Solandt and Magladery (1940). Muscle temperature must remain constant during the assessment of the action of a drug on fibrillating muscle.

(7) Muscle atrophy in man can be detected before the onset of fibrillation. Thus atrophy is not solely dependent on "overwork" due to fibrillation.

(8) The discrepant findings in the literature have been discussed in the light of the metabolic factor affecting fibrillation of denervation.

Acknowledgments

Our thanks are due to Professor W. E. Le Gros Clark, F.R.S., and H. J. Seddon, F.R.C.S., for their help and encouragement. Mr. P. Peade, of the Department of Anatomy, University of Oxford, rendered much valuable technical assistance; to him we also offer our sincere thanks. The expenses of this investigation were defrayed by a grant to one of us (G. W.) by the Medical Research Council, which is gratefully acknowledged. (B. F.) was in receipt of a personal grant from the Carnegie Research Fund and (R. E. P.) from the Nuffield Fluid Research Fund; these are also gratefully acknowledged.

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NEUROSIS AND SEXUALITY

BY

ELIOT SLATER

(RECEIVED 31ST JANUARY, 1945)

Introduction

NEUROTIC men have been found to differ from normal men in a number of particulars, in the frequency with which they show qualitative findings and in the degree to which they show quantitative findings. In addition they have been found to be abnormally heterogeneous. In respect of graded characters we may expect them at times to vary from normals at both ends of the scale. If we choose a single criterion for the distinguishing of neurotics from normals, we cannot expect it to be of value over more than part of the range; it may be useful in distinguishing some neurotics but not all. For instance, Rees and Eysenck (1945) have shown that an index relating stature to transverse chest diameter (1) exhibited greater variability in the neurotic than in the normal, (2) was associated at one end of the scale with symptoms of anxiety, depression, effort syndrome, etc., (3) at the other extreme was associated with hysterical symptoms. Attention is rightly being paid to the physical correlates of psychological qualities. For physical characteristics are often capable of preciser definition and more exact measurement than any psychological characteristic with which they may be associated; and the discovery of an association between a physical and mental trait may give us a clue to causal relationship. For this purpose it is desirable to examine the neurotic and the normal subject particularly in respect of those physical and psychological qualities where deviations might be expected to have an effect in reducing good health, adaptability, or emotional stability.

In the psycho-analytic theory of the development of the neuroses it has always been maintained that the eventual neurotic adaptation of the adult is determined by disturbances of one kind or another in early psycho-sexual development. This theory has been supported by a wealth of individual observations, but by no general evidence that the sexual activity of neurotics differs in any way from that of the normal. A certain amount of evidence of this kind has, however, been forthcoming from other sources. Billings *et al.* (1943) made a comparison between 100 U.S. Army psychiatric patients and 100 enlisted men. Of the psychiatric patients 54 were neurotic or showed psychopathic personality, while the remainder showed schizophrenic or affective symptoms, or were feeble-minded. They state that 44 per cent. more of the enlisted men than of the patients showed an average sexual development, 30 per cent. had regular girl friends 41 per cent. more had intentions to marry. Absolute figures are not given. Also in the U.S.A. Steinberg and Wittman (1943) have reported on their findings on 267 men from the Services. Of these 158 formed a control group, 22 were predominantly neurotic, and 87 predominantly psychotic. As tested by the Elgin developmental history, the psychotic subjects (of whom 76 per cent. were schizophrenic) made a less satisfactory early sex adjustment than the controls, but the difference between the neurotic patients and the controls was negligible; the same was largely true for adult sex adjustment. As far as the female is concerned, it is worth noting that Bernard (1935), working with a group of married couples, found significant positive correlation coefficients between (1) marital dissatisfaction of wife and neuroticism of wife, (2) marital dissatisfaction of husband and neuroticism of wife, and (3) days since last coitus and neuroticism of wife; there was, however, no significant correlation between days since last coitus and neuroticism of husband. It cannot be asserted that any of this evidence is very precise or takes us very far.

In a previous communication (Slater, 1943), dealing with the statistical analysis of the clinical records of two thousand neurotic soldiers, it was observed that a considerable proportion (45 per cent.) had led lives in which sexual activity had played a surprisingly minor rôle. In these men " sexual relations began at a late age, occurred with unusual rarity and were associated with little interest or satisfaction." The question whether the sexual activity of an individual patient was to be regarded as inadequate or normal in degree was decided by the doctor in charge of the case. No doubt standards of judgment varied from clinician to clinician. But as each doctor would be consistent with his own standard, the division of the case material into two classes in this way does represent a real though crude distinction. From here on the one group will be referred to as " inadequate " and the other as " average " in respect of sex life. The statistical material was analysed, using this criterion for classification, but consideration of the findings was postponed as it was hoped to combine the data

with those obtained from a biochemical and genetical investigation which was then in progress. Unfortunately, these latter investigations had to be interrupted, and are not likely to be completed in the near future. The results of the statistical investigation are, however, so interesting that it is thought that they merit publication on their own account. In the table below are listed all and only those findings in which there was a statistically significant difference between the frequencies observed in the two groups of men with " inadequate " and with " average " sex lives.[1]

Results

Several interesting points appear from the table. The first is that the group of under-sexed men are clearly a more abnormal group than the others. In them, with few exceptions, the positive psychiatric findings are more frequent. The exceptions are themselves interesting. Head injury is more frequent in the group of men with " average " sexuality; it is a finding which has already been shown (Slater, 1943) to be associated with lesser rather than greater degrees of constitutional predisposition. The amnesic symptoms are also more frequent in the averagely sexed group; these symptoms are particularly frequent among men who break down in battle (Sargant and Slater, 1941). The only abnormal personality trait which is more frequent among the averagely sexed are the qualities of impulsiveness or aggressiveness. This can be related to the finding by Denis Hill that patients suffering from the clinical electro-encephalographic syndrome which he has termed " dysrhythmic aggressive behaviour " (Hill, 1944) are very commonly troubled by a quite excessive libide (personal communication).

The second point of interest is that where a graded characteristic is being considered, the sexually inadequate tend to be found in excess at one or other extreme. Compared with men of average sexuality, they are more likely to have either a poor or a good school record, to be either of poor or of good intelligence, and to be relatively infrequently average in these respects. They are more likely to have an asthenic or a pyknic physique and are less likely to be of athletic habitus. Exactly parallel findings have recently been reported from comparisons of neurotics as a whole with normal controls. Eysenck (1943) has found that neurotics were less frequently average in respect of intelligence, and Rees and Eysenck (1945) have found that they were less frequently average in respect of bodily habitus than were the normal men with whom they were compared.

Observation	Sex life				C.R.	P less than
	Inadequate		Average			
	N.	Per cent.	N.	Per cent.		
Childhood:						
Neurosis *	487	33·4	701	28·7	—	—
Neurotic traits ..	—	38·3	—	37·1	—	—
Normal ..	—	28·3	—	34·2	2·2	0·05
Home life:						
Unsatisfactory * ..	419	24·3	637	14·4	3·9	0·001
School record:						
Poor * ..	499	31·0	690	26·6	—	—
Average ..	—	50·1	—	57·9	2·6	0·01
Good ..	—	18·7	—	15·5	—	—
Occupation:						
Semi-skilled manual *	501	35·9	695	44·8	3·1	0·01
Sedentary *	—	13·3	—	6·5	3·8	0·001
Army rank:						
N.C.O's. * ..	516	15·6	717	20·9	2·4	0·02
Previously:						
Head injury	516	11·3	717	20·5	4·5	0·001
Military stress ..	—	38·8	—	50·9	4·3	0·001
Precipitation	—	41·4	—	54·4	4·6	0·001
Bodily habitus:						
Asthenic *	353	53·3	538	44·5	—	—
Athletic * ..	—	23·5	—	34·4	3·5	0·001
Pyknic ..	—	23·3	—	21·1	—	—
Intelligence						
Poor * ..	456	34·1	658	26·9	—	—
Average * ..	—	53·4	—	64·1	3·6	0·001
Good * ..	—	12·5	—	9·0	—	—
Personality						
Anxious ..	516	25·3	717	19·1	2·5	0·02
Hysterical ..	—	16·7	—	11·7	2·5	0·02
Impulsive, aggressive ..	—	15·5	—	21·7	2·8	0·01
Unsociable ..	—	40·2	—	30·1	3·7	0·001
Anergic ..	—	29·1	—	22·4	2·7	0·01
Generally abnormal ..	—	51·6	—	41·4	3·5	0·001
Symptom:						
Amnesia *	516	13·6	717	19·2	2·8	0·01
Outcome:						
Unimproved *	516	26·0	717	18·8	2·9	0·01

Table showing Frequencies of Various Observations among Neurotic Military Patients, classified by Sex Life.
N.=number of persons on which relevant observations made.
C.R.=critical ratio, i.e. ration of difference between percentages to its standard error.
P.=probability of such a ratio being obtained by chance.
*=comparable finding made when patients classified by degree of military stress undergone (see Table II, Slater, 1943).

The third point is that the table that is published here bears a very remarkable resemblance to Table II in my previous communication (Slater, 1943), where the same clinical material was classified by degree of military stress undergone. All the findings marked by an asterisk in the present table were found to be associated with degree of military stress, and in every case the association is of the

[1] It is to be noted that there was a small but significant difference between the mean ages of the " inadequate " and " average " groups. This indicated that the clinicians, in forming their judgment, had not been able to allow sufficiently for the effects of age. For the young must of necessity have had as a rule a less abundant sex life than their seniors. It was therefore thought desirable to make allowance for this adventitious factor. The necessary corrections for age, which all proved to be very small, have therefore been made throughout the table, a process which though laborious was quite simple as the material had been classified by age groups as well as by sex life in the primary card counts.

same kind; i.e. a finding which is more frequent among the sexually inadequate than among the average, is more frequent among those who have not undergone military stress than among those who have. Breakdown in the absence of military stress and inadequate sexuality are themselves positively associated; and it might be supposed that this association might by itself explain their common associations with other psychiatric findings. This suggestion, however, cannot be supported. For, if these associations are expressed in the form of correlation coefficients, the coefficients are all of the order of 0·2; that is, they are so low that partial correlation coefficients corrected for their communalities would be very little smaller. The several associations of these two findings, degree of stress undergone and degree of sexual activity, must be regarded as having independent validity.

Discussion

We must conclude therefore that the factors which predispose to neurotic breakdown are related to those which tend to bring about a reduction of normal sexuality. This is entirely consonant with the hypothesis of Freud. There are, however, other ways of looking at the matter, two of which particularly suggest themselves. We might suppose that the relationship between tendency to neurosis and tendency to sexual inhibition might be in either of two directions. It is possible that the listlessness and inadequacy of personality, which shows itself in so many neurotics, was also expressed to some extent in their sex lives. This would imply that impoverished sexuality was an almost accidental by-product of the general tendency to failure and defeat. On the other hand, it is possible that constitutional factors, such as minor degrees of endocrine insufficiency of various kinds, which result in deficient sexuality, were so impairing the personality as to increase the liability to nervous breakdown. Both hypotheses seem plausible; there is no reason why both should not be to some extent true; and we have no present evidence which would lead us to favour either at the expense of the other. Nevertheless, it may be pointed out that the former hypothesis does not lend itself to translation into any very precise terms, and cannot be subjected to any simple test. The second hypothesis suggests the advisability of investigating the endocrine balance of neurotics, and even provides a hint that the results of such an investigation might eventually prove to be not only of ætiological but also of therapeutic significance.

Summary

The clinical records of 1,233 neurotic soldiers were classified by the degree of sexual activity they had shown and the frequencies of various psychiatric findings were analysed in the two groups of the sexually inadequate and the sexually average. Sexual inadequacy was found to have significant associations with neurosis in childhood, unsatisfactoriness of home life, school record, occupation, rank in Army, past head injury, degree of military stress undergone, bodily habitus, intelligence, various personality traits, amnesic symptoms, outcome of illness. In nearly every case sexual inadequacy was found to be positively associated with a higher frequency of the abnormal finding. Where a graded characteristic is in question, such as in intelligence and bodily habitus, sexual inadequacy was more frequent at the extremes of the distribution. The findings are closely paralleled by a similar analysis of the same clinical material by degree of military stress undergone. It is concluded that the factors which tend to bring about neurotic breakdown and those which predispose to sexual inadequacy are related. Various hypothetical bases for this observation are discussed. One which appears to be particularly favourable for further investigation suggests that the common factors are to be sought in an imbalance of the endocrine system.

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AN EXPLORATORY STUDY OF MENTAL ORGANIZATION IN SENILITY

BY

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(RECEIVED 9TH FEBRUARY, 1945)

Introduction

THE fact that in the last few decades the proportion of the old and senile has been growing at an increasing rate and that this trend shows every likelihood of continuing has centred recent scientific interest on the psychological changes which take place in senility. As Dayton has shown (1940), Senile Dementia has now displaced Schizophrenia as the most common psychosis requiring hospital treatment, and many social problems are raised by the increasing average age of the population.

Studies have been made of the ability of old people, shown by tests of intelligence, memory, motor co-ordination, imagination, and other functions. The work of Wechsler (1942), Miles (1933), Thorndike (1928), Sorenson (1933), Ruch (1923) and others has left little doubt that mental ability decreases with increasing age once adulthood has been reached. It has also been shown that this decrease takes place at different rates for different tests, and a ratio of the relatively slowly decreasing Vocabulary Test to the relatively quickly decreasing abstract type of intelligence test has been made into an index of senile deterioration (Babcock, 1941; Gilbert, 1935).

While these facts may be regarded as established, further points of interest remain to be investigated. It appears possible that, in addition to the quantitative differences observed between the abilities of the old and the young, qualitative differences also might be observed. Qualitative differences and changes which Piaget considers to contribute to the growth of intelligence may also become evident in its decline. An investigation into this problem has been reported elsewhere (Eysenck, 1945).

Another problem will be dealt with in the present paper. None of the studies so far mentioned deal with the organization of the various abilities involved in the tests used; yet it seems reasonable to suppose that differences in organization should become apparent with advancing age. For instance, it has been reported that much higher intercorrelations than are found for adults are usually observed for children. On the other hand, group factors often assume increased importance in the older subjects (Cattell, 1943). These facts make it necessary to extend the factorial study of mental abilities to senile groups in an attempt to throw some light on the organization of mental functions in old age.

Method

(*a*) *Population.*—The population used in this experiment consisted of well over 100 male patients with Senile Dementia at the Tooting Bec Hospital. This number was reduced during the course of the experiment through death, discharge from the hospital, etc., to below 100. Approximately 10 per cent. were illiterate. These various factors reduced the number of patients included in the main experiment to 75. Twenty of these had been labourers, handymen, carmen, and unskilled factory workers; 20 had been highly skilled workers (compositors, instrument makers, engravers, etc.), had been in business for themselves or had managed shops for others; the remaining 35 were either unclassifiable or had been employed in jobs roughly intermediate between the other two groups as regards the skill required in their previous profession. The average age was $73{\cdot}4 \pm 6{\cdot}5$.

(*b*) *Administration.*—Altogether 20 tests were given, which are described in Section (*c*). The tests were given individually in two sittings separated by about six weeks. Testing time was 59 minutes on the average for the first sitting and 51 minutes on the average for the second sitting. Wide individual variations were, of course, encountered. The whole battery of tests was repeated after four months. The patients were tested at the same time of day as before. On the repetition the average time for the first sitting was 48 minutes (with two tests omitted); the second sitting 44 minutes. Thus each of the patients was tested individually for an average of approximately three and a half hours.

Eighty-four patients altogether were tested twice on all the tests, except for the fact that 9 patients were illiterate and were incapable of doing two of the tests requiring writing. Test-retest reliabilities were calculated on the 84 for all the tests except the two writing tests; correlations were calculated only for the 75 patients who had done all the tests including the writing tests.

(*c*) *Tests.*

Psychological Test Battery (given on first sitting):

(1) *Orientation.*—The first test was presented as a necessary formality. The patient was asked his full name, his age, date of birth, the current day's date, the approximate hour of the day, and the letter and number of the ward he was in. The total possible score on this test is 5 points.

(2) *Progressive Matrices.*—The individual board form

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† The Laboratory and the research on ageing were financed from the grants to the Club for Research on Ageing by Lord Nuffield and the Nuffield Foundation, to whom the Club and the author express their gratitude. They thank also the London County Council for all the facilities given at the Tooting Bec Hospital for the work.

of Raven's Progressive Matrices was given (1940). The board form consists of only Sets " A " and " B " of the total Matrix Test. The total possible score on this test is 24 points.

(3) *Digits Forward.*—This test was given in the usual way, starting with two digits and continuing till three successive attempts at any particular number of digits had failed.

(4) *Digits Backward.*—This was given in exactly the same way as Test (3). In both tests the highest number of digits correctly repeated or reversed constituted the score.

(5) *Immediate Memory.*—The story " Three Houses on Fire " (Burt, 1933) was read to the patient after he had been warned that he would be asked to tell as much as he could remember of it. His free memory was supplemented by questions, as the senile patients' spontaneous response did not do justice to their memory. This story aroused much emotional reaction, the incident described becoming identified with the London blitz, and another, more neutral, story was substituted in the second testing. Spontaneous memory and answers to questions counted equally towards total score. Maximum score on both stories was 20 points.

(6) *Fluency.*—The patient was asked to name as many kinds of (*a*) animals, (*b*) birds, and (*c*) flowers as he could in one minute each. Scores on these three subtests were combined to give the total number of objects named during three minutes. Timing started when the patient pronounced the first name in each series.

(7) *Days of the Week.*—The patient was asked to say the days of the week as quickly as he could, the time taken being his score.

(8) *Days of the Week Backward.*—The patient was asked to say the days of the week backward as quickly as he could, the time taken being his score.

(9) *Months of the Year.*—The patient was asked to say the months of the year as quickly as he could, the time taken being his score.

(10) *Months of the Year Backward.*—The patient was asked to say the months of the year backward as quickly as he could, the time taken being his score.

(11) *Delayed Recall.*—At the end of Test (5) the patient was told that he would be required to retell the story later on. He was now asked to do so, scoring being exactly as in Test (5).

(12) *Vocabulary.*—The Mill Hill Vocabulary Test was used, the patient being required to supply definitions for a series of 44 words increasing in difficulty. The number of correct definitions constituted his score.

(13) *Writing Name.*—The patient was asked to sign his name with a soft pencil on unlined paper. The time taken was recorded and scored as the rate per 10 letters.

(14) *Writing from Dictation.*—The patient was asked how long he had been in the hospital and then told to write " I have been here . . . years (or months)." This was also scored as the rate per 10 letters.

(15) *Incidental Memory.*—The patient was asked to recall as many of the different problems he had done as possible.

The following two tests were used on the first testing but were then dropped from the battery.

(i) *Colours.*—The four colours given by Burt (1933) were presented to the patient and he was asked to name them. He was also asked to say which he liked best, second best, and so on.

(ii) *Porteus Mazes.*—This test was rejected because it was too difficult for many of the subjects, and because vision plays too large a part in it.

Motor Test Battery (given on second sitting):

(16) *Strength of Grip: Dynamometer.*—This test was given to the patient with his elbow resting on the table and the other hand on his knees. Two tests were done for each hand in alternation. The average of these four scores, in kilograms, constitutes the score of the patient.

(17) *Speed of Tapping.*—The patient stood with his left hand gripping the back of a chair and was asked to tap with his other hand as quickly as he could from one metal plate to another for 30 seconds. After 30 seconds' rest the test was repeated. The number of double taps on both trials together constitutes his score.

(18) *Arm Ergograph.*—The patient's arm above the elbow was strapped to an arm rest while his hand lay on a metal disc which he was required to press against the resistance of a spring. The extent of each stroke was recorded on a kymograph. The patient was told to keep time with a metronome and continued the test for two minutes. A two minutes' rest was interpolated, and another two minutes' trial performed. The score used was the average length of stroke expressed in terms of eighths of an inch.

(19) *Ink Blot.*—To allow the patient to recover from the exertion of the previous test a non-motor test was introduced here. He was shown a coloured ink blot and asked to pick out as many different things in it as he could. He was stopped after 2½ minutes. The number of items given constituted his score. (This test was not included in the factor analysis because of its low test-retest reliability, $r=0{\cdot}46$, and because of its extremely low correlations with the other tests.)

(20) *Steadiness.*—The patient was required to hold a stylus inside a hole in a metal plate without touching the side. Holes of different diameter were used (the test is illustrated in Whipple (1924, p. 157)). Each trial continued for 15 seconds. The score was determined by a combined measure of size of hole and number of touches, the higher the score the better the performance on this test.

The following two tests were used on the first testing but were then dropped from the battery.

(*a*) *V-groove.*—In this test the patient was required to trace a path between two gradually narrowing lines. This test was dropped as a measure of steadiness in favour of Test (20) after the first testing had been completed. In order to obtain a reliability coefficient for Test (20) it was given twice on the occasion of the second testing, once at the beginning, and again at the end of the sitting.

(*b*) *Persistence.*—The patient was asked to hold a weight in his outstretched hand for as long as he could, the time he held up the weight constituting his score. The test was repeated after two minutes' rest, and proved so unreliable that no further use was made of the data.

Results

In Table I are given the average performances on the first and second testing, the S.D.'s of the scores, the average improvement of the second testing expressed as a percentage of the score on the first testing, and the reliability coefficients for the various tests. The reliabilities of the combined (averaged) scores on the two testings, as estimated by the Spearman-Brown Prophecy Formula are also given. Except where otherwise stated these combined scores were used in all the calculations reported in this paper.

Perhaps the first point that will be noticed in the table is the fact that the S.D.'s are relatively large. This would naturally be expected from the test scores of subjects so divergent in cultural background, education, professional status, and deterioration.

Of the 18 tests where average improvement could be calculated, 5 showed deterioration. Of the 4 motor tests included 3 show a decrease on the second testing, probably indicating that the passage of time, even a few months, causes some deterioration in physical strength in subjects of so advanced an age. As will be seen later, the motor test scores correlate more strongly with age than do the rest.

TABLE I

Test	Average, 1st testing	S.D., 1st testing	Average 2nd testing	S.D. 2nd testing	Percent. improvement	Reliability coefficient	Combined reliability coefficient
(3) Digits forward	5·9	1·1	5·7	1·1	−3	0·59	0·74
(4) Digits backward	3·7	1·1	3·9	1·0	5	0·69	0·82
(7) Days forward	3·5	1·6	3·1	2·1	11	0·90	0·95
(8) Days backward	11·3	26·4	9·7	21·2	14	0·51	0·68
(9) Months forward	7·5	5·2	6·6	4·3	12	0·83	0·91
(10) Months backward ..	51·0	50·6	39·0	34·5	24	0·68	0·81
(13) Writing name	17·5	12·6	16·9	15·2	3	0·96	0·98
(14) Writing dictation ..	18·3	13·1	17·2	11·9	6	0·89	0·94
(19) Ink blot	4·7	2·8	5·8	3·1	25	0·46	0·63
* (5) Immediate memory ..	4·5	2·9	4·3	3·1	—	—	—
*(11) Delayed recall	4·8	3·5	5·9	4·1	—	—	—
(15) Incidental memory ..	3·8	3·0	4·3	3·7	13	0·78	0·88
(1) Orientation	3·8	1·1	3·8	1·1	0	0·89	0·94
(12) Vocabulary	18·0	7·1	17·9	6·8	−1	0·95	0·97
(6) Fluency	23·5	8·4	25·7	9·5	9	0·82	0·90
(20) Steadiness..	49·7	39·5	64·3	52·2	29	0·80	0·89
(17) Tapping	88·9	22·6	86·1	20·8	−3	0·82	0·90
(16) Dynamometer	19·9	6·2	17·9	6·4	−10	0·89	0·94
(18) Ergograph	12·1	3·6	11·6	3·8	−4	0·86	0·92
(2) Matrix	14·7	2·9	15·1	3·1	3	0·49	0·66

* In the case of these two tests the scores on the second testing only were used. As different stories were used on the first and second testing the reliability coefficient would not be comparable to the others.

The fourth motor test, steadiness, however, shows the largest amount of improvement. As has been explained, this particular test was substituted for another after the first testing, and was given to the subjects at the beginning and at the end of the second testing. It is natural, therefore, that the practice effect should come out most strongly: in every other case there was a time interval of three or four months between the first and second testing, whereas not more than 25 minutes elapsed between the two testings in the case of the steadiness test. The general tendency for the scores of the other tests to improve on the second testing is undoubtedly due chiefly to the practice effect and in a few cases to the better rapport secured.

On the whole the reliability coefficients are rather higher than might have been expected with these subjects. When the combined scores of the first and second testing are used, a fair degree of reliability is achieved.

In Table II are shown the product-moment correlations run between 20 variables for 75 subjects. Of the 190 correlations only 9 are negative, none significantly so. With the level of significance according to Fisher's method being 0·22 ($p=0·05$), it is seen that 101 of the correlations reach or exceed it. At the $p=0·01$ level of significant, $r=0·29$, and 69 of the correlations reach or exceed it.

The correlations seem to fall into three fairly well defined groups, and Burt's group factor method (1940) was accordingly used to factor analyse the table. The great majority of the tests fit in well with these group factors; four tests, whose attribution is doubtful, have the group factor saturation shown in brackets, viz. vocabulary, fluency, matrix, and age. The factor saturations appear in Table III.

The interpretation of the three group factors presents little difficulty. The majority of the tests making up Group Factor I are tests involving speed. The inclusion of the digits tests, however, makes it difficult to consider this factor as one purely of speed. It is of interest to note that days forward,

TABLE II

	(3)	(4)	(7)	(8)	(9)	(10)	(13)	(14)	(5)	(11)	(15)	(1)	(12)	(6)	(20)	(17)	(16)	(18)	(2)	Age
(3) Digits forward	—	0·43	0·41	0·35	0·37	0·33	0·30	0·34	0·18	0·17	0·11	0·03	0·31	0·17	0·11	0·02	0·01	0·28	0·20	0·09
(4) Digits backward		—	0·77	0·37	0·23	0·51	0·36	0·35	0·36	0·27	0·25	0·22	0·44	0·25	0·18	0·27	0·06	0·06	0·16	0·14
(7) Days forward			—	0·34	0·77	0·28	0·41	0·33	0·29	0·24	0·00	0·04	0·09	0·31	−0·14	0·17	0·28	0·20	0·05	0·02
(8) Days backward				—	0·23	0·47	0·30	0·41	0·11	0·15	0·13	0·00	0·20	0·10	0·05	0·01	0·01	0·12	0·14	0·20
(9) Months forward					—	0·36	0·37	0·44	0·26	0·25	0·11	0·18	0·25	0·34	−0·17	0·15	0·19	0·14	−0·03	−0·15
(10) Months backward ..						—	0·27	0·45	0·36	0·38	0·34	0·44	0·46	0·28	0·15	0·28	0·13	0·40	0·14	0·11
(13) Writing name							—	0·68	0·04	0·09	0·05	0·06	0·35	0·26	0·13	0·43	0·14	0·14	0·03	0·13
(14) Writing dictation ..								—	0·17	0·28	0·27	0·23	0·64	0·34	0·02	0·33	0·30	0·15	0·09	0·12
(5) Immediate memory ..									—	0·78	0·46	0·37	0·40	0·33	−0·07	0·12	0·28	0·15	0·15	0·20
(11) Delayed recall										—	0·65	0·54	0·40	0·45	0·04	0·11	0·21	0·24	0·21	0·17
(15) Incidental memory ..											—	0·59	0·35	0·45	0·22	0·23	0·18	0·22	0·31	−0·01
(1) Orientation												—	0·29	0·38	0·31	0·26	0·18	0·29	0·17	−0·11
(12) Vocabulary													—	0·43	0·14	0·18	0·03	0·22	0·26	0·05
(6) Fluency														—	−0·04	0·32	0·25	0·31	0·32	−0·02
(20) Steadiness															—	0·11	0·37	0·26	0·31	0·13
(17) Tapping																—	0·40	0·44	0·15	0·12
(16) Dynamometer																	—	0·60	0·38	0·22
(18) Ergograph																		—	0·20	0·11
(2) Matrix																			—	0·04
Age																				—

months forward, and writing name have in every case higher saturations than their companion test, where something a little more out of the ordinary is required, namely, days backward, months backward, and writing dictation. In the light of these six tests it would appear feasible to say that this factor goes some way towards measuring the natural speed of the subject.

TABLE III

Test	General Factor	Group Factor I	Group Factor II	Group Factor III
(3) Digits forward ..	0·33	0·45	—	—
(4) Digits backward ..	0·52	0·45	—	—
(7) Days forward ..	0·30	0·77	—	—
(8) Days backward ..	0·24	0·51	—	—
(9) Months forward ..	0·30	0·57	—	—
(10) Months backward	0·67	0·25	—	—
(13) Writing name ..	0·36	0·47	—	—
(14) Writing dictation ..	0·57	0·41	—	—
(5) Immediate memory..	0·51	—	0·45	—
(11) Delayed recall ..	0·55	—	0·76	—
(15) Incidental memory..	0·47	—	0·66	—
(1) Orientation	0·45	—	0·43	—
(12) Vocabulary	0·71	—	(0·01)	—
(6) Fluency ..	0·62	—	(0·17)	—
(20) Steadiness	0·14	—	—	0·46
(17) Tapping ..	0·45	—	—	0·27
(16) Dynamometer ..	0·35	—	—	0·88
(18) Ergograph	0·45	—	—	0·47
(2) Matrix ..	0·34	—	—	(0·25)
Age ..	0·15	—	—	(0·16)
Variance ..	0·20	0·10	0·07	0·06

Group Factor II is mainly concerned with tests of memory; even the bracketed tests (vocabulary and fluency) are at least in part a matter of (long term) memory.

Group Factor III is made up of tests of physical strength and ability. Age, while its membership in this group factor is somewhat doubtful, shows a tendency to be more strongly related to physical strength than to the other groups of more strictly psychological tests. The one exception to this is the matrix, the aptness of whose inclusion here is also in doubt. From this analysis it would appear that this test is rather more in agreement with the motor powers than with the mental powers in the case of these aged subjects. It must be borne in mind, however, that any definite conclusions about the relation between age and physical and mental powers could not be looked for from a population whose ages were so closely grouped together, and whose mental and physical deterioration were from observation comparatively unrelated to age. An investigation with a wider range of middle-aged and old people might show more clearly the exact relation between advancing age and deterioration in mental and physical abilities.

The general factor presents certain difficulties in interpretation. The factor saturations of such tests as have been used in previous factorial studies of children or adults below the age of this test group present a distinctly different pattern from that found here. If the present general factor were identical with that found in the usual type of analysis we should expect the matrix test to show a higher saturation than 0·34, and we should expect the motor tests to show little or no appreciable saturation with this factor. It seems reasonable to conclude that the unequal deterioration of various kinds of ability in old age leads to a different pattern of organization from that found in normal adults. In understanding the changes which take place in the mental life of the aged knowledge of the organization of their abilities as well as of the comparative strength of these abilities is of obvious importance.

Cattell (1943) draws a distinction between two kinds of adult mental capacity which he calls " fluid " and " crystallized."

" Fluid ability has the character of a purely general ability to discriminate and perceive relations between any fundaments, new or old. It increases until adolescence and then slowly declines. . . . Crystallized ability consists of discriminatory habits long established in a particular field, originally through the operation of fluid ability, but no longer requiring insightful perception for their successful operation. Intelligence tests test at all ages the combined resultants of fluid and crystallized ability, but in childhood the first is predominant whereas in adult life, owing to the recession of fluid ability, the peaks of performance are determined more by the crystallized abilities."

If this hypothesis is true, we should expect tests of fluid ability, such as the matrix, to have very high factor saturations in a study of adolescents and young people generally but to give low factor saturations in extreme old age; while, conversely, crystallized abilities would give comparatively high factor saturations in old age. This is precisely what we do find in this analysis, and we may, therefore, consider that this analysis gives a certain measure of support to the theory set forth by Cattell.

This opinion is not in accord with Cattell's own view about the consequences of his theory. He writes:

" . . . if the crystallized abilities are, as it were, a dead coral formation revealing by its outlines the limits of growth of the original living tissue, these crystallized abilities will show approximately the same intercorrelations as the original fluid abilities."

He goes on to say that " we should expect the intercorrelations to approximate the childhood values but to be systematically lower," and gives several reasons to justify his argument.

This argument is possibly over-simplified, and does not do justice to the facts. What we are dealing with are abilities, both crystallized and fluid, which have deteriorated at different rates; they would show approximately the same correlations as the childhood values, on a rather lower plane, only if the *rate of deterioration* could be shown to be perfectly correlated with the *extent of the original abilities*. But there is no evidence in favour of this assumption; what evidence there is argues rather for a comparatively slight correlation, or none at all. Thus for a young person, a measure of fluid ability like the matrix would intercorrelate with his other abilities very highly, and have a high factor-saturation for "g"; for a senile person, there is no reason to suppose that what remains of his fluid ability, as measured by the matrix, should intercorrelate very highly with his crystallized ability, as measured by the vocabulary test, for instance. Proof of this assertion is given below. The same argument holds when we are dealing with tests involving both fluid and crystallized ability in varying proportions.

Further support for this view comes from a consideration of the scores of seniles on some of the tests used and a comparison of these scores with those of normal adult groups. The best representative of "crystallized' ability is probably the vocabulary test. On this test the seniles' average score is well above the 14-year-old level for normal subjects. The range between + and −1 S.D. about the mean extends from the average verbal ability of normal adults to that of 9 to 10-year-old children. Compared with normal adults their average score is around the 25th percentile.

The most representative test of "fluid" ability perhaps is the matrix test, on which the average performance of the senile group is almost identical with that of 8-year-old children. In terms of the normal population the seniles would come between the 3rd and 4th percentiles.

A comparison between the results of these two tests shows clearly the differential deterioration of the abilities concerned. It is impossible, of course, to tell from our data whether, and if so to what extent, the "crystallized" ability has decreased; in other words, it is not claimed that a measure of absolute deterioration can be given from the data.

For normal adults the progressive matrices test and the Mill Hill vocabulary test show a correlation in the neighbourhood of 0·6. The correlation found between the scores on these two tests for the senile patients is only 0·26. Even when this correlation is corrected for attenuation by the use of Hull's formula (1928, p. 243) it rises to only 0·33, and is thus very significantly lower than even the uncorrected correlation obtaining for normal adults. This would seem to indicate that not only do the abilities measured by these two tests deteriorate at unequal rates, but also that the rate at which the abstract type of ability measured by the matrix test deteriorates is not highly correlated with the original amount of that ability possessed by the subjects.

On the whole, the conclusions drawn from the comparison of the results of the matrix and vocabulary tests for the seniles agree with those drawn by Cleveland and Dysinger (1944).

"The majority of institutionalized senile patients are unable to sort objects on the basis of conceptual or abstract principles, but are limited to concrete bases. . . . (They) were able to respond to many of the verbal items in the Wechsler-Bellevue Scale on an apparently abstract verbal level, but were unable to sort objects on an abstract or conceptual basis. This appears to be due to the fact that a patient may use what seems to be an abstract verbal concept with much more restricted meaning."

On digits forward the seniles' average score of 5·9 is roughly equal to the norm for 8-year-olds. On the original Binet immediate memory test their average of 4·7 appears to be slightly higher than the 8-year-old level. Owing to the different method employed it is not possible to equate the results with any great exactitude.

Wechsler's paper on "Intellectual Changes with Age" (1941) includes an interesting graph, based on Galton's data, showing the mean grip in pounds (right hand) for males from age 5 to 80. The average dynamometer score for the seniles (right hand) transformed into pounds is about 44, which corresponds approximately to that found for the 15-year-olds, and is considerably lower than Galton found for 80-year-olds. The difference of time, nearly 70 years, between Galton's work and this investigation, is yet another factor making a direct comparison difficult; the type of dynamometer used, the technique, as well as the likelihood that this particular sample of seniles is not truly representative (being composed of a large number of men accustomed to hard physical labour, who probably lack their usual exercise and diet in hospital), all contribute to the uncertainty of any conclusions that might be drawn. It appears safe to say that there is some deterioration demonstrated in this test of dynamometer grip, but it seems to be somewhat less pronounced than the deterioration observed in the mental tests.

In an effort to make further deductions from the data collected, the 75 subjects were divided into three groups: 20 whose previous profession had been definitely superior to the average of the whole group; 20 whose previous profession had been definitely inferior to the average; and the remaining 35 whose profession was either uncertain or about the average level for the whole group. In Table IV average scores for the top and bottom groups are shown, the difference between their scores, the S.D. for the whole group of 75, and the difference expressed in terms of these S.Ds. The figures in the last column, it should be noted, are not critical ratios; nor are they in any sense estimates of the statistical significance of the differences; they are intended rather to make possible a comparison of the differences observed between the two groups in the various tests.

TABLE IV

Test	Average score superior group	Average score inferior group	Difference	S.D. of total group	Difference ÷ S.D. of total group
(3) Digits forward	5·9	5·8	0·1	1·1	0·09
(4) Digits backward	4·1	3·6	0·5	1·0	0·50
(7) Days forward	2·6	3·4	0·8	2·6	0·31
(8) Days backward	5·6	26·3	20·7	24·7	0·83
(9) Months forward	6·5	7·5	1·0	5·5	0·18
(10) Months backward	53·3	90·2	36·9	68·8	0·54
(13) Writing name	11·4	18·2	6·8	9·5	0·72
(14) Writing dictation	11·5	24·8	13·3	12·7	1·05
(5) Immediate memory	5·9	4·2	1·7	3·2	0·53
(11) Delayed recall	7·6	5·5	2·1	4·3	0·49
(15) Incidental memory	5·2	3·5	1·7	3·2	0·53
(1) Orientation	4·2	3·8	0·4	1·0	0·40
(12) Vocabulary	22·2	14·7	7·5	6·6	1·14
(6) Fluency	25·3	22·0	3·3	8·8	0·37
(20) Steadiness	75·7	46·9	28·8	43·1	0·67
(17) Tapping	97·3	87·0	10·3	19·6	0·52
(16) Dynamometer	21·1	18·0	3·1	6·1	0·51
(18) Ergograph	12·8	11·3	1·5	3·6	0·42
(2) Matrix	15·8	14·6	1·2	2·4	0·50
Age	75·2	72·2	3·0	6·6	0·45

The first point of interest is that on every single test the superior group scores more highly than the inferior. It is perhaps surprising that on the tests of physical strength also this superiority shows itself. As we have seen, there is a slight tendency for age to correlate with these motor tests (i.e. the younger subjects tend to do rather better on the whole), and what little difference there is in age between the two groups favours the inferior. Possibly part of the explanation is the better nutrition the superior group have enjoyed before hospital, and perhaps in it owing to their larger supply of spending money for extras at the patients' canteen.

The largest differences, as shown by the figures in the last column of the table, are in the vocabulary and the writing from dictation tests. That is probably what one might have anticipated from the way in which the two groups were selected. It is interesting to note that the digits backward, days backward, and months backward tests in each case show a larger difference between the two groups than do the digits forward, days forward, and months forward tests. The matrix test shows comparatively little discrimination between the two groups, less than the purely physical tests on the average.

In Table V are shown the varying distributions of the vocabulary scores for the two groups, as compared with a normal adult group, and with the total group of 75 seniles.

This shows clearly the difference in the results of the vocabulary test of the two groups. Only 10 per cent. of the inferior group reach the "average," whereas nearly half the superior group are "average" or better. It will be noted that even the superior group have 55 per cent. "definitely below average." Judging from the occupations followed it would appear improbable that such a high proportion of them in their prime were unable to achieve at least average verbal rating. However, it is well known that one of the chief difficulties of all vocabulary tests is that the words employed in common speech change from generation to generation, and even granting the possibility that the vocabulary grows until about the age of 40, a whole generation has intervened since the vocabulary of these subjects was laid down.

TABLE V

Verbal ability	Percentage of each group			
	Normal adults	Whole group seniles	Superior seniles	Inferior seniles
Superior ..	5	0	0	0
Above average ..	20	8	20	0
Average ..	50	27	25	10
Below average ..	20	47	55	55
Defective ..	5	18	0	35

These results cover the tests included in the final battery. It may be of interest to mention briefly some results of the colour test which was originally included in the psychological test battery but which was later removed.

In all, 110 subjects did the test with sufficient understanding for their results to be used. Of this number 19 saw one or more of the colours, red, green, blue, and yellow, imperfectly. In 10 cases green was seen as blue of some sort, and upon questioning they asserted that there was no green there at all. Red was seen as mauve by two subjects and another two saw it as brownish. These findings suggest that perhaps colour vision declines in old age, a conclusion in line with the results of Smith (1943).

In addition to naming the colours, the subjects were asked to rank them in order of preference. The 19 who were unable to see all four colours correctly were omitted in this calculation, leaving 91 ranking orders. Blue was liked best, getting an average score of 1·55, while yellow was liked least, with an average score of 3·37. Red and green were almost equally popular, with an average score of 2·71 and 2·37 respectively. In H. J. Eysenck's work on colour preferences (1941) he found blue the favourite and yellow the most disliked of these particular four colours; but the positions of red and green were reversed. As the shades used in these researches were not identical, however, too much importance should not be attributed to this reversal.

The average intercorrelation of the rankings in the group of seniles was 0·32, which is almost identical with that found by H. J. Eysenck in his study ($r=0·28$).

Summary and Conclusions

Twenty psychological tests were given individually to 84 senile dementia patients and repeated after four months, total testing time per patient being approximately three-and-a-half hours. The average age of the patients was $73·4 \pm 6·5$ years. Test-retest reliabilities were established for the tests used, and intercorrelations run between the tests. A factorial analysis was carried out on these intercorrelations, in an attempt to throw some light on the type of mental organization to be found in senility. Comparisons were made between the scores of the patients on various tests and the scores of normal adults and children on these tests. A comparison was also made between the scores on the tests of a " superior " and an " inferior " group of seniles, as determined by the level of the skill required in the work they had been doing in the course of their lives.

With few exceptions, the reliabilities found for the tests were remarkably high, thus demonstrating that it is quite feasible to apply psychological tests of the type used even to senile dementia patients. A slight improvement was found for most of the tests from first to second testing, which was probably due to practice effects and " test sophistication."

The factor analysis resulted in a factor-pattern which accounted for 43 per cent. of the variance, a general factor contributing 20 per cent. to the variance, and three group-factors, identified as being concerned mainly with speed, memory, and physical strength, contributing 10, 7, and 6 per cent. respectively.

The general factor presented a picture of the mental organization of the patients which differed greatly from that found in normal adults. An attempt was made to account for this difference in terms of the theory of " fluid " and " crystallized " ability.

The level of the senile patients on the progressive matrices test of abstract non-verbal ability approximated that of 8-year-old children; similarly, their performance on the digits repetition test was similar to that of 8-year-old children. On the vocabulary test, however, the seniles scored well above the 14-year-old level. The correlation between matrix and vocabulary for the senile patients was shown to be very significantly lower than the same correlation for normal adults. These facts were also brought into relation with the theory of " fluid " and " crystallized " ability.

The comparison of the superior and the inferior senile patients showed the superior group consistently scoring at a higher level than the inferior group, even on tests of physical strength. The degree of superiority, however, varied within wide limits.

The results allow the following conclusions :

(*a*) Mental ability in the seniles tested has considerably deteriorated.

(*b*) This deterioration affects most strongly tests involving abstract reasoning ability (" fluid " ability).

(*c*) This deterioration affects least tests involving a mere reactivation of past experiences and knowledge (" crystallized " ability).

(*d*) Due to the differential deterioration in the abilities of senile patients, the organization of these abilities, as compared with normal adults, becomes profoundly altered.

The author is indebted to Dr. P. Turnbull, Medical Superintendent of the Tooting Bec Hospital, for the facilities given during the research work; and to Drs. Aubrey Lewis, Wm. Stephenson and P. E. Vernon for their many helpful suggestions. The author also wants to thank Mr. H. Halstead and Mr. J. C. Raven for supplying her with certain unpublished data.

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CYCLIC OCULOMOTOR PALSY

BY

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THERE have been described about thirty cases of this interesting phenomenon. Axenfeld and Schürenberg (1901) have their names attached to the condition in the Continental literature; they called it "Cyclic Congenital Oculomotor Disease". Fuchs, however, described a case much earlier (1893). The clinical picture is quite different from recurrent oculomotor palsy. The cause is still a matter of conjecture as no case has been examined post-mortem.

Essentially the clinical picture is a rhythmic, or cyclic palsy of the muscles moving the eye and usually also of the levator muscle of the upper lid associated with dilatation of the pupil during the stage of full paresis and ptosis, and contraction of the pupil as the lid rises and the muscles of the globe recover part of their power. The condition is unilateral and may be acquired, or congenital. The movements are quite involuntary and the rhythmic alteration of tone in the paralysed muscles goes on during sleep and waking. When there is intermittent ptosis the condition is quite striking. The fourth and sixth nerves are much less commonly paralysed than the third, or part of the third, nerve. When the ptosis is absent and the extraocular muscle palsy is slight the pupil only may show the rhythmic movement. The mild cases of cyclic oculomotor palsy without ptosis may therefore be readily overlooked.

Case History

C.H. aged eight years. Spontaneous delivery. The right eyelid has appeared abnormal since birth. There is almost a complete ptosis, but every few minutes throughout the day and night the right eye opens involuntarily to a normal extent for a period of about thirty seconds. This movement is quite involuntary and cannot be influenced in any way by the child's own action. On timing the active (lid raised) and passive (ptosed lid) stages over fifteen minutes, the average interval between active stages is one-and-a-half minutes and the lid remains up for thirty seconds. The longest interval between phases was two minutes, and the shortest, one minute. In the ptosed or passive stage the palpebral fissure is three mms. wide (Fig. 3) and it rises in jerks until the fissure is of normal width (Fig. 2). The mother states that when the child is sitting quiet the lid does not rise so often. Sometimes the lid does not rise so completely. Voluntary movement of the eyes did not seem to influence the rate of lid rising. Prostigmine had no effect.

There is a divergent squint of the right eye, the corneal margin being one mm. from the outer canthus as can be seen in Fig. 1. There is a slight adduction of the globe during the active, or lid raised stage as can be seen in Fig. 2. There is no voluntary vertical movement of the right eye, and no downward movement on adduction. The only voluntary movement is slight abduction.

Fig. 3 shows the passive stage of ptosis. The pupil during the passive stage is dilated in the right eye—diameter four mms. (Fig. 1) and reacts very poorly to light and accommodation, and to pain and lid closure stimuli. It is regular. During the active phase the pupil becomes equal to the normal fellow pupil and reacts

FIG. 1.

normally to light and accommodation. The refraction in the passive stage in the right eye is $\frac{+0{\cdot}50}{-1{\cdot}00/180^\circ}$ and this becomes $\frac{-0{\cdot}50}{-1{\cdot}50/180^\circ}$ when the lid rises. With $-1{\cdot}50/180^\circ$, vision is improved in the right eye from

FIG. 2.

6/36 to 6/18 Snellen. Vision in the left eye is 6/9 Snellen, there being a small error of hypermetropic astigmatism. Cocaine 2 per cent. dilates the pupil widely as does also Homatropine 1 per cent.; Eserine ½ per cent. and

FIG. 3.

Pilocarpin 2 per cent. produce contraction of the pupil. Examination with the corneal microscope reveals no structural abnormality in the anterior segment of the eye. No other neurological abnormality was found. Stereoscopic X-ray examination of the skull revealed a thickening of the right sphenoidal ridge and this was reported to be of congenital origin. The child looks healthy and is well grown for her age and the Wasserman reaction is negative. The family history is negative and the child has had no serious illness.

Discussion

The clinical picture of these rare palsies varies somewhat in the literature of the thirty cases reported. Any oculomotor nerve may be involved, or

any branch. The lid paralysis occurred in two-thirds of the cases. The internal rectus is very commonly involved and the superior oblique, rather uncommonly. There is rarely involvement of other cranial nerves, the fifth, seventh, and ninth. Only one bilateral case is described and the lids were involved alternately, one eye being in the active phase, while the other was in the palsied, or passive phase. The sixth nerve usually escapes. Contraction of the dilated pupil during the active phase is a constant feature with or without activity of the internal rectus. Occasionally there is nystagmus in one, or both eyes. Nearly all cases show amblyopia of some degree in the affected eye. The classical description of Axenfeld and Schürenberg has had to be modified as more cases came to be reported and it was seen that the condition need not be congenital. Greeves (1913) reported a case in a girl aged seven years, after measles. Salus (1912) described a case aged five years with suppurating submaxillary glands which required operation, the condition of unilateral cyclic oculomotor palsy supervening thereafter. At the age of twenty years this patient still had intermittent discharge from these glands. Bielschowsky (1929), who described four cases out of a series of 1,700 cases of ocular palsy, showed that the intermittent or cyclic character of the palsy may occur some time after the onset of palsy. Later Bielschowsky (1935) collected the reports of thirty-two cases of cyclic oculomotor paralysis and of these he had seen ten cases himself. He was convinced that there were slight or rudimentary instances of the phenomenon and that if such cases were included that the condition would be found not to be quite so rare as is supposed. The alternate contraction and dilatation of the pupil is the only feature common to all the cases described. There is great variation in the degree of paralysis of the extraocular muscles and levator palpebral muscle on the affected side. No one extraocular muscle is constantly implicated in the palsy, though one or more supplied by the third nerve are usually involved. In one-third of the cases Bielschowsky says the upper lid is not involved and there is no intermittent ptosis. The elevator and depressor muscles of the eyeball never participate in alternating spasm and relaxation. Mild cases therefore may show only the phenomenon of alternating relaxation and contraction of the apparently paretic pupil with slight paralysis of one or more of the extraocular muscles or the levator muscle of the lid. As such they may be missed or reported as a peculiar pupil-phenomenon with a third nerve palsy. Here the intermittent, or cyclic part of the clinical picture is limited to the pupil movement and Bielschowsky thinks these cases should be included in the group of cyclic oculomotor palsies. In half the cases the condition is acquired in early childhood; the others are congenital. One single case is reported as beginning at the age of seventeen. Bielschowsky attributes the cyclic behaviour of the muscles to varying vasomotor conditions in the region of the third nerve nucleus.

Salus (1912) thought that this cyclic oculomotor palsy was due to aberration of regenerating nerve fibres as is considered to be the cause of "mitbewegung", or, as it is called in this country, "the jaw-winking phenomenon of Marcus Gunn", where the palsied lid rises with the action of the pterygoids. Bielschowsky when speaking of the pseudo Græfe phenomenon thinks that this is due to regenerating fibres from the nucleus finding their way into the wrong paths. The movements of cyclic oculomotor palsy, however, are not initiated by voluntary movement as in the "jaw-winking" and the pseudo Græfe phenomena. The essential feature of cyclic palsy is that it is automatic and rhythmic. Bielschowsky thought that the lesion was in the neighbourhood of the third nucleus and that vasomotor influences produced the rhythmicity. It may be that the blood supply is impaired by disease or injury, or congenital defect and that there is a "storage" as it were of sub-minimal stimuli in the damaged nucleus, which when they become adequate lead to periodic discharge along the spared, or partially spared, paths. The influence of excitement or anxiety in increasing the rate of the cycle might be explained on this vascular basis. When the child was sitting quietly reading the rate of the cycle was less.

In our case there was a thickening of the sphenoidal ridge on that side which was considered probably to be congenital. The child was too young for arteriography. In adults this might prove of value in revealing a possible abnormal blood supply, but it is likely that the solution still awaits the evidences of post-mortem examination.

Summary

1. A case of cyclic oculomotor palsy as described, of which over thirty cases have appeared in the literature.

2. No report on autopsy has been made in any of these cases.

3. The interest of this oculomotor palsy lies in its cyclic, or rhythmic and automatic character.

The explanations are necessarily conjectural, but it seems possible that damage has occurred to the nucleus, or its efferent paths congenitally, or as a result of disease. There may be some "storage" of sub-threshold stimuli in the remaining effective part of the nucleus and these stimuli when they reach adequate intensity, overflow periodically to produce the lid rising and associated changes of the active phase of the cycle.

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SOME OBSERVATIONS ON THE EFFECT OF CENTRIFUGAL FORCE IN MAN

BY

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(RECEIVED JUNE, 1945)

ANY flight manœuvre, whether simple or complex, imposes varying degrees or types of acceleration upon the aircraft and the crew. Centripetal acceleration is developed when an aircraft is moving in a curved path, and its value is directly proportional to the square of the angular velocity. For a constant indicated air-speed, the centripetal acceleration is inversely proportional to the radius of curvature of the flight path. It follows from Newton's First Law of Motion that a force must be acting upon the aircraft and directed normally towards the centre of curvature, but on account of inertia, the aircraft resists this force; the line of action of this resistance is opposite to the centripetal force, is therefore directed away from the centre and is termed centrifugal force. If the pilot be regarded as an inanimate part of the aircraft, it is convenient to think in terms of this centrifugal force, which presses him into his seat and is responsible for the physiological effects.

It is also convenient to express the value of centrifugal force (G) as multiples of gravity by the following formula:

$$G = \frac{V^2}{15r} \cdot g \text{ (gravity units)}$$

where V = indicated speed in m.p.h.
r = radius of turn in feet.

In practice, this formula is of little value since the force acting upon the pilot is the resultant of the centrifugal force and gravity, and is measured directly by an accelerometer.

The sensations attendant upon exposure to centrifugal forces of any magnitude have been described graphically and in some detail by Rook and Dawson (1938), Livingston (1939), Diringshofen (1938 and 1939). The latter author and his collaborators have investigated many aspects of the problem and as a result of their basic researches, it can be stated that the more severe effects are due to a fall in blood pressure in the carotid arteries. This causes the loss of vision known as " Blacking Out " amongst fighter pilots and also the loss of consciousness, which, however, is infrequent in occurrence. It follows that any practical method, by which the fall in blood pressure can be prevented, also aids the pilot in retaining vision. The importance of this to any pilot, during combat, need not be stressed. The fall in blood pressure in the carotid arteries is due to the line of action of the force passing vertically through the spine of a pilot in the sitting position. Not only has the heart to pump blood against this force, but the cardiac output also falls due to reduction in venous return and pooling of the blood in the abdomen and lower limbs.

An opportunity has been afforded to one subject (W.K.S.) to make frequent observations of the subjective effects, and an analysis of some of the objective effects, of centrifugal force in flight. This was achieved by the use of cine photography in a two-seater aircraft. Approximately 50 subjects have been observed by this method, but the following account deals principally with the correlation of the objective and subjective effects of the subject (W.K.S.).

The effects of these forces may be classified in three main phases: first, the general sensations of the apparent increase in weight of the body; secondly, the loss of vision or " blacking-out "; thirdly, the loss of consciousness which is infrequent and is usually preceded by a period of " black-out ".

General Sensations

Somatic sensations are principally dependant upon the effective increase in weight. At G loadings of over 5, movements of limbs in the plane of the centrifugal force either become impossible or extremely difficult according to the physical build of the individual. Movements in a plane at right angles to the force were still possible at these loadings but were comparatively slow and awkward in unsupported limbs. There may also be a significant increase in the latent period which may be partly central and partly peripheral in origin (Burmeister, 1939). Care must be taken in the interpretation of such experimental results since the line of axis of some of the muscles concerned in the movement may be in the plane of centrifugal force. Further common symptoms of the increase in weight were a downward drag of the facial tissues and the " concertina sensation " of the trunk. Objectively, the effects of these were easily seen in the deepening o

the naso-labial folds, nasal ridges becoming more pronounced and in the development of subocular pouches, the lower lids sometimes showing a marked eversion (Figs. 1 and 2). If the erect posture were maintained, not only did the soft tissues become moulded but there also occurred an apparent de-

FIG. 1.—Subject seated in rear cockpit of Battle. Kollsman Accelerometer recording approximately 5 G.

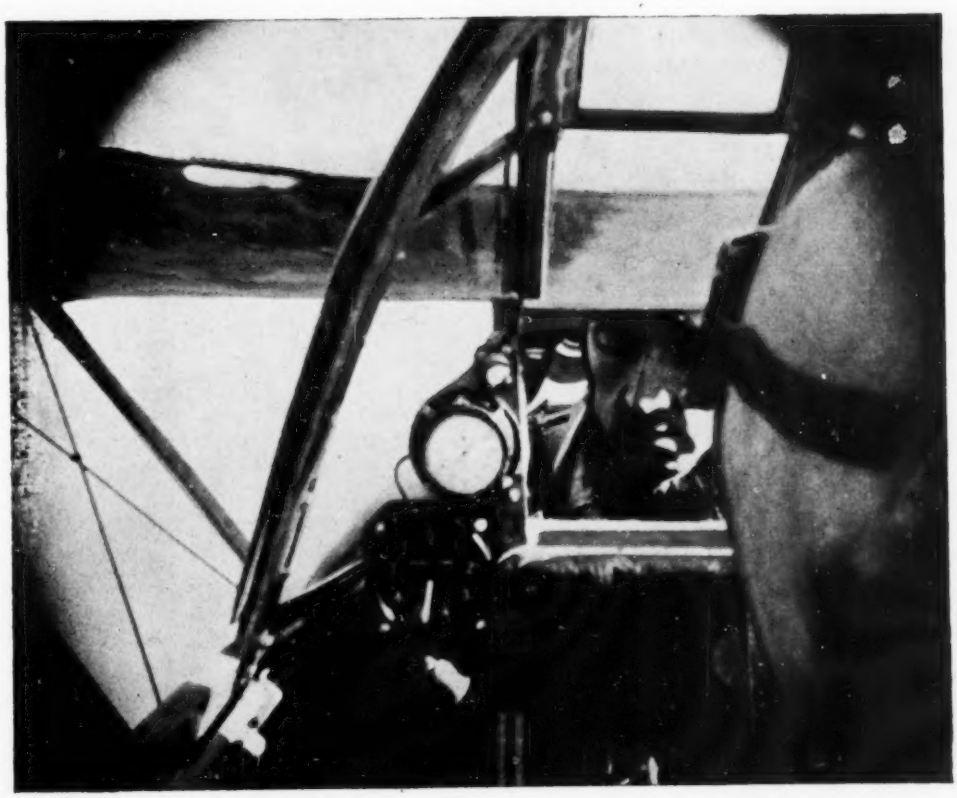


FIG. 2.—Subject in Gladiator. Kollsman Accelerometer recording approximately 5 G.

crease in the height of the trunk of 1–2 inches, and if these effects were marked, the subject appeared to have acquired new features. Under heavy G, whether vision is affected or not, most subjects appear to have delayed cerebration, to be slightly dazed as if their spatial and temporal orientation are impaired.

Visual Impairment—Blacking-out

The above reactions are of secondary importance to the disturbance of vision or of consciousness. It was found necessary, early in the course of the research, to standardize the recognition of the end-point of the visual loss. Consequently a test-type card with letters of standard dimensions was hung in the cockpit before the observer, and above the camera level. Thus for all comparative work, the abolition of contrast between letters and background was taken as the commencement of the " black-out " period. At no time did distant vision remain after this had occurred, the amaurosis being complete. There was, however, considerable difficulty in analysing the sequence of events. When the test-type card was taken as the visual stimulus, the usual degrees of impairment could be differentiated easily. Slight " veiling " could be distinguished as a mere greying of the white background, the black letters still being distinct. Severe " veiling " could be differentiated as a further stage, when the card would become almost black and yet the central letters still be distinguished as a form of tunnel vision. The end-point was reached when the letters had disappeared, the general sensation being that of blackness. These stages usually succeeded one another, although, not infrequently, the first two stages seemed to be omitted, the onset of the " black-out " being extremely sudden. These experimental observations can be compared to the sequence of events in fighter aircraft. In the turret of a two-seater fighter, it was observed that according to the final value and rate of onset of G, distant vision became blurred and was lost before near vision. Objects subtending a small angle at the retina, such as details of the ground and distant aircraft, definitely disappeared before turret details such as the ring and spot of the reflector sight. " Veiling " of vision was generally associated also with darkening of the clouds, and blurring of cockpit detail. The final end-point of vision was of course similar in that everything became black as if the sensation of light were lost. Thus, little information could be derived from an analysis of the amaurosis, for although in most cases it was similar to the action of a diaphragm, yet often the classical German description of " Vorhang " applied. The onset was so rapid that the order of impairment of the upper and lower segments of the visual field could not be determined. Occasionally an amaurosis of rapid onset appeared not to give a sensation of " blacking-out " but of " whiting-out "—as if light were still appreciated. This has also been experienced by some pilots and is not uncommon. The whole of the visual field was affected; at no time was there any hemianopic or scotomatous impairment. Finally the return of vision was rapid. Intermediate stages could not be differentiated, nor were residual visual symptoms ever experienced.

Oculomotor Reactions

It has been pointed out before that general somatic sensation is not subjectively greatly impaired when " blacking-out " takes place and that co-ordinated movements are still possible. In general, the integrity of the external oculomotor muscles was found to be maintained during the period of " black-out " and full and co-ordinated ocular movements were possible in every direction. There did not appear to be any significant prolongation of the time taken to complete these movements when " blacked-out ", as compared to the time taken normally. Fixity of gaze appeared to be a constant feature during the " black-out " period. This can be ascribed to the subject concentrating on timing as accurately as possible the duration of the visual loss. Three experiments were carried out during which the subject was " blacked-out " with his head turned strongly to the right but with gaze fixated on the test-type card. There was no deviation of gaze from the point of fixation even in an amaurosis lasting for 11–12 seconds.

During the course of the experiments, a left-sided esophoria had been observed under high loadings (5 G or over). The degree of imbalance, though slight, was definite, occurred usually in the left eye and appeared to be peculiar to the individual (Fig. 3). It was most often associated with the development of the amaurosis, but also occurred before visual impairment was experienced, and also at high G when the subject was protected from the circulatory effects. Once developed, the esophoria remained throughout the period of high G, and on one occasion progressed to a stage almost of skew deviation. On another occasion the eyes were voluntarily moved whilst the esophoria was present. The visual axes were then seen to be readjusted—the imbalance disappearing during the movement and developing again in the period of rest.

Nystagmoid movements were never recorded, but frequently at high values of G there developed a considerable degree of ptosis of the right upper eyelid, not unlike a unilateral Horner's Syndrome. This was frequently associated with " blacking-out ", but also developed apart from visual impairment (Fig. 4). There was never any paresis of the facial musculature.

Sensations associated with Loss of Vision

Other sensory functions did not appear to be markedly impaired. At no time was there any actual loss of hearing as reported by Rook and Dawson (1938). If auditory acuity became impaired there was always an association with pressure effects in the middle ear sustained during the preliminary dive. There was never any actual nausea, sickness, giddiness, or tinnitus during the application of high acceleration. Sensory somatic functions were also

FIG. 3.—Esophoria of left eye in severe black-out. Acceleration value 4·4 G. (Maximum—5 G.)

FIG. 4.—Ptosis of upper eyelids in blacked-out subject. Acceleration value 5·8 G.

relatively unaffected—even in an amaurosis lasting as long as 12 seconds. Posture was normally maintained; limb positions were appreciated and movements of small amplitude were executed with ease. The sense of smell was found to be present when the subject was " blacked-out ". Oil of cloves vapour delivered through a nasal catheter during a prolonged " black-out " was appreciated. Pain was never experienced. Headache of frontal type was occasionally experienced by the pilot, but its origin could not be dissociated from sinus pressure. An interesting sensation which was frequently recorded, was the occurrence of paræsthesia in the legs. This was of a tingling or burning character—localized to the calves and lateral aspects of the legs. It only occurred under heavy G loadings and was unrelated to the actual " black-out ", being experienced by the pilot who had complete retention of vision and also by the subject who had visual loss. These symptoms were absent when the circulation in the lower limbs was arrested by means of cuffs, and therefore may be due to hydrostatic distension of the vessels. However, analysis of sensations incurred with heavy loadings must at best be rudimentary—apart from the dominating sensation of the loss of vision. This caused some psychological upset, evidenced usually by apprehension often associated with an unpleasant feeling in the abdomen. Most subjects experienced apprehension in their first few " black-outs ", and it was noteworthy how disconcerting a " black-out " could be to pilots with many hundreds and thousands of flying hours to their credit, but who, owing to the nature of their work, had not been subjected to more than 3½–4 G for a considerable time. However, apprehension disappeared as the subject became accustomed to " blacking-out ".

Removal of Acceleration

The removal of G did not cause any symptoms. Just as the general increase in the weight of the limbs must cause some peripheral sensorimotor disturbance, likewise the removal of the force may have similar effects on the kinæsthetic judgment. It was frequently observed, for example, that the subject's conception of when the G was finally removed and his subsequent behaviour, often coincided with an accelerometer reading of 2 G. In two of fifty subjects, when the aircraft was resuming level flight and the G falling off, there was distinct spatial disorientation associated with a sense of diving steeply, almost as if the aircraft were turning on its back. Dizziness, nausea, and profuse sweating were also noticed. A complete " black-out " at 5¼ G in one of these subjects only resulted in slight discomfort, but again spatial disorientation was experienced, during removal of the G, and caused the subject to lean back in an effort to overcome the sensation of falling forward. Although postural improvement allowed the subject to sustain 5·5 G without visual symptoms, it did not alleviate the dizziness and associated effects. It is interesting to note that this observer is prone to air-sickness,

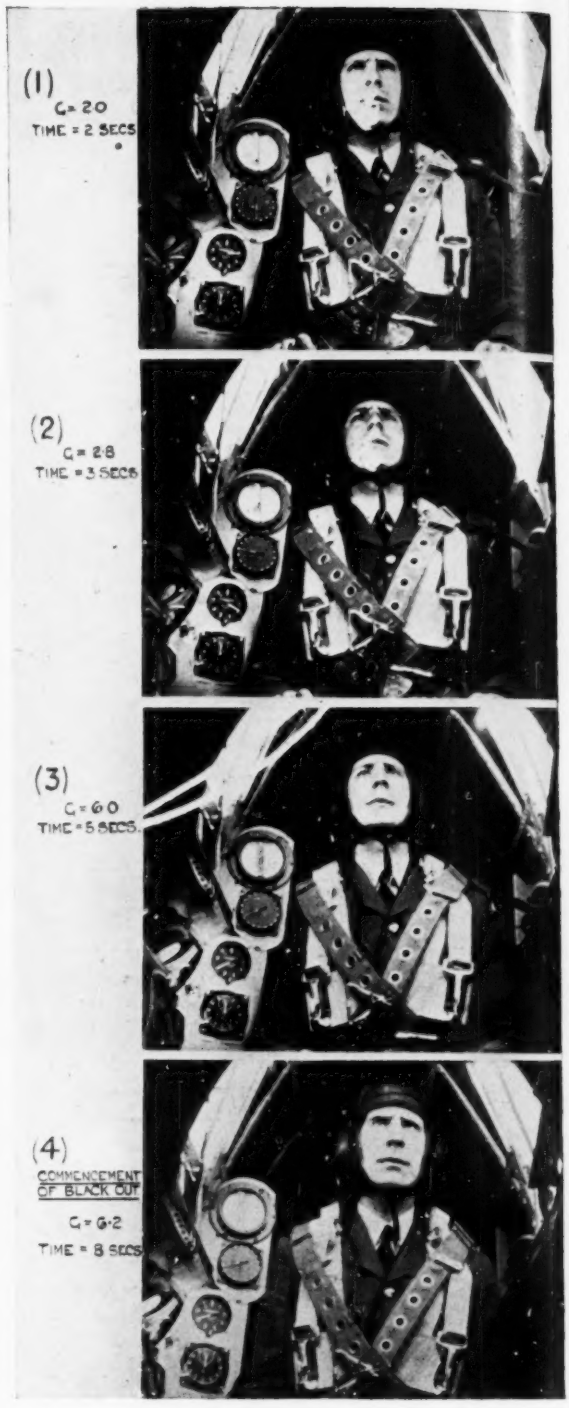


FIG. 5.—Onset and development of unconsciousness.

but at the time of these tests adaptation had taken place.

After Effects of Blacking-Out

Immediate sequelæ were not marked for W.K.S., but occasionally pilots reported some slight loss of spatial orientation in that, after " blacking-out " severely, a further slow circuit would have to be made in order to establish their surroundings. On completion of any flight, the most marked effect was the feeling of undue tiredness, lethargy, and fatigue.

FIG. 5.—Onset and development of unconsciousness.

This was particularly noted in the lower limbs—presumably because they were the first members to be exercised. In addition to this, however, power of concentration would be definitely diminished after 3–4 consecutive " black-outs "; memory also seemed to be impaired, and the subject frequently felt depressed and unsociable in the evening. Occasionally there was some disturbance of the sleep rhythm—early morning awakening being a feature. These effects were much more marked at the commencement of the period of research, but continued in some measure or other throughout the whole period—the fatigue and lethargy being especially persistent. No permanent impairment of eyesight occurred. A fact worthy of further investigation emerged in that after any period of intensive flying, the subject, who had slight heterophoria, definitely experienced headaches associated with occasional double vision. These symptoms were not experienced during periods in which no flying took place and had never been noted by the subject before. Pilots and subjects complained

occasionally of backaches due to muscular strain. X-rays revealed no abnormalities of the vertebral column. There were no effects upon the cardio-vascular or nervous systems and no tendency to develop varicosities in veins of legs.

Unconsciousness

Total loss and impairment of consciousness of various degrees was experienced by W.K.S. on several occasions. The introduction of the term impairment is, of course, open to some objections, as it is based on the recording of motor reactions. It is firmly believed, however, that there is a gradient in the loss of function of nervous tissue which ranges from some lengthening of reaction time at sub-threshold values of G, through the stage of visual symptoms, to the total abolition of all motor and sensory functions. This final stage is always associated with true amnesia and loss of consciousness. The criterion for cortical impairment could only be derived from the correlation of subjective effects with objective or automatic photographic recording, since no performance tests or other investigations could be applied.

Complete unconsciousness was only deemed to have been present when the following events were recorded: (1) Definite alteration in posture and motor reactions. (2) Loss of general sensibility and muscle tone. (3) Amnesia. (4) Subjective sensations greater than those experienced in simple " blacking-out ". Summaries of two experiments illustrate these points. It must again be emphasized that this subjective and retrospective analysis must be regarded as being coloured with considerable psychological disturbance.

Complete " black-out " was achieved rapidly in one experimental flight. The subject then found himself lying on the floor of the cockpit in front of the camera, having fallen completely out of the harness, which at that time was a simple gunner's waist belt. There was no recollection of striking the floor, although both face and arms were considerably bruised. Sound was first appreciated, then pain and posture, and finally vision, though this may be partly due to the fact that illumination in the well of the aircraft was of low intensity. The principal emotion was fear, but nevertheless general psychological upset and disorientation were present —for example, fluid which had poured from the inverted nasal sinuses under the acceleration was initially thought to be cerebro-spinal fluid.

A second experiment is illustrated in Figs. 5 and 6. On both these occasions it did appear as if some degree of consciousness were recovered before full return of vision.

Impairment of consciousness was said to exist when there was: (1) No marked alteration in general posture but some loss in the motor reactions of the limbs and muscle tone. (2) Subjective sensations not experienced in " blacking-out ". The difference between impairment and complete unconsciousness is more academic than practical since control of the aircraft would have been lost in either case, if the subject had been piloting. Impairment included some loss of somatic sensation particularly of the arms, associated with loss of muscular power, but amnesia was absent in that the subject did not recognize any marked change in his environment after vision returned.

The recognition of a period of unconsciousness is rarely attained unless it produces a marked change in environment. This, of course, is always produced in single-seater aircraft, due to loss of control; but under the experimental conditions, in which the subject, knowing that the procedure under investigation might produce unconsciousness, and taking the precaution of adjusting the safety harness tightly, recognition of unconsciousness could not always be definite. It has been found that impairment might occur without the subject being aware of more than a very severe " black-out ". In one case, marked alteration in posture occurred, a subject falling forward and striking his nose on the intercommunication tubing without even being aware that he had " blacked-out ". This was never experienced by W.K.S., a preceding stage of visual loss always occurring.

Loss of posture under centrifugal force is magnified by the increase in weight of the various body components. Any loss of muscular power may be greatly enhanced by the mechanical effects of the G upon the part affected. One has also to be cautious of interpreting loss of posture on non-medical or inexperienced observers. There may be some instinct present to yield to the acceleration by bending forwards particularly when " blacked-out ". This can be complicated by the fact that any given pilot who may be undergoing acceleration tests for medical categorization may confuse the terminology " black-out " with loss of consciousness.

Apart from nausea and a general feeling of malaise, the symptoms varied somewhat with the experimental procedure. For example, when the jugular veins were compressed visual impairment under G was associated with sensations referred to the head, with clonic spasm of the hands, and precordial pain. A beating sensation in ears and head was also not uncommon. Clonic twitching of hands, and individual digits was often experienced on recovering consciousness and sometimes after severe " black-outs ", particularly when associated with experiments on the effects of G and anoxia. In one of 50 subjects, however, clonic twitching involving the upper limbs and facial musculature of gross amplitude was observed whilst recovery of consciousness was taking place. The exposure field of the camera unfortunately did not include the lower limbs, but Diringshofen (1941) has stated that generalized convulsions are not uncommon during or after consciousness. Incontinence of urine was noted on one occasion.

A factor of almost equal importance to the actual loss of consciousness was the disturbance in spatial and temporal orientation which was always present in some measure on return to consciousness. It is not known whether this was an intrinsic physiological

effect, but it has been observed to last from 10–20 second after the erect posture has been regained. The subject was always at a loss to account for changes in orientation on recovery; mental processes were markedly impaired during this period. He might hear the pilot faintly over the intercommunication system, but neither had the will-power nor the understanding to answer. Fully co-ordinated action, unless of an automatic nature, appears to be impossible.

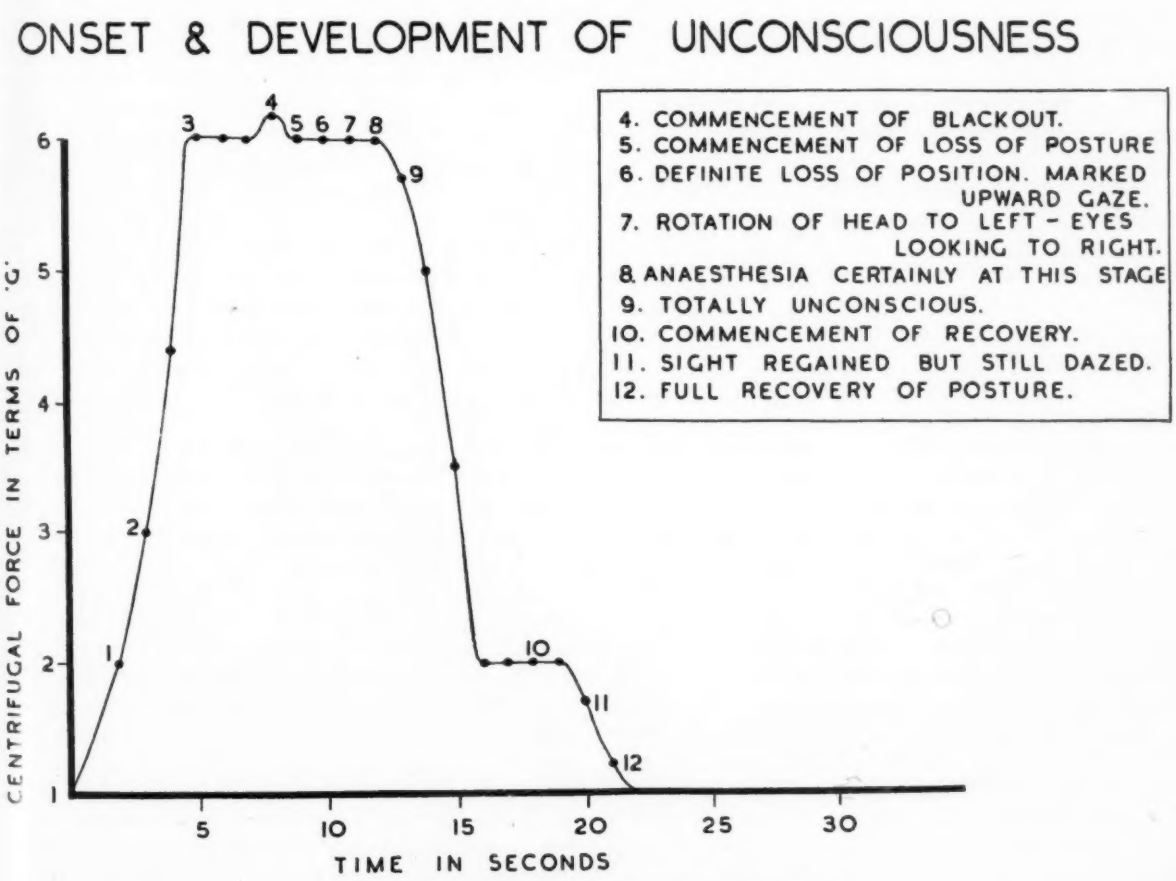
After effects were similar to, but more marked than in any threshold run. One definite feature has been noticed after unconsciousness, a definite lowering of morale or will-power, normally expressed as a disinclination to undergo further immediate experimentations. If this were necessary, considerable apprehension existed, which was reflected in undue tensing of the muscles, and holding of the breath whilst undergoing the ensuing acceleration.

Physiological Judgment of Value of Acceleration

The capacity of any one individual to estimate the value of the acceleration may be limited. This is in accordance with the conception of the bombardment of the kinæsthetic sensorium by a flood of stimuli under the great increase in weight. Thus it is difficult to estimate the value of any amount greater than 4 G to within 1 G, especially if the observer be occupied in piloting. It can be stated, however, that 9 G is very different from 6 G, and 6 G from 4 G. A resistant subject is generally aware of the onset of heavy centrifugal force at about 4 G.

Discussion

There is ample and well-established evidence that the loss of function due to centrifugal force is related to the circulatory effects. It is beyond the scope of this article to attempt any review of the literature, but it is an accepted fact that the visual impairment is primarily retinal in localization. The visual impairment can be considered as a rise in the subjective threshold of the retina and is similar in characteristics to the rise in threshold caused by obstruction of the retinal circulation by external pressure, Craik and Vernon (1941). The latency, between the appearance of ocular symptoms and disturbance of consciousness, is generally ascribed to the intra-ocular tension, of between 25–30 mm. of mercury, which causes an initial effective decrease in the blood pressure in the central retinal artery as compared to the intracranial pressure. Thus the retina is rendered anoxæmic before the central nervous system (Andina, 1937). There has always been some doubt as to the probable degree of impairment of cortical function when a pilot is " blacked out ". Further, it has been thought that the visual impair-


ONSET & DEVELOPMENT OF UNCONSCIOUSNESS
4. COMMENCEMENT OF BLACKOUT.
5. COMMENCEMENT OF LOSS OF POSTURE
6. DEFINITE LOSS OF POSITION. MARKED UPWARD GAZE.
7. ROTATION OF HEAD TO LEFT - EYES LOOKING TO RIGHT.
8. ANAESTHESIA CERTAINLY AT THIS STAGE
9. TOTALLY UNCONSCIOUS.
10. COMMENCEMENT OF RECOVERY.
11. SIGHT REGAINED BUT STILL DAZED.
12. FULL RECOVERY OF POSTURE.
CENTRIFUGAL FORCE IN TERMS OF 'G'
TIME IN SECONDS
1 2 3 4 5 6 7 8 9 10 11 12
1 2 3 4 5 6
5 10 15 20 25 30

Numbers correspond with photographs in Fig. 5.

FIG. 6.

ment may be caused either by pressure upon the optic tracts, due to the increase in weight of the frontal lobes or to occlusion of the posterior cerebral artery, as it crosses the tentorium, by the posterior lobes. The first hypothesis can be dismissed, since it is most unlikely that shearing stresses could be developed in the optic chiasma or tracts, of sufficient degree to produce blockage of the transmission of nervous impulses; and it is known that hydrostatic pressure on the tracts could not produce the visual impairment (Grundfest, 1936). It is, however, difficult to exclude the second hypothesis on evidence at present available. At values of G which do not produce visual symptoms, no reduction in general efficiency can be demonstrated in pilots flying single-seater aircraft, and no impairment of nervous function can be revealed in practical or performance tests which involve flying an aircraft, or in associated duties. However, if low values of G be very frequently repeated in one flight, fatigue does develop during the flight, is experienced during the post-flight period and may not disappear without rest or sleep. If the flying duties involve continual exposure to low values of G over a period of weeks or months, this type of fatigue may become associated with symptoms of anxiety. At values of G which cause visual impairment, it has been demonstrated that the whole field of vision is affected although the degree of impairment may vary for central or peripheral vision. Owing to the nature of the research, perimetry could not be attempted, but it did appear evident that scotomatous or hemianopic defects were never present. The value of G which, if maintained for 3–5 seconds, results in total visual loss, is termed the " blacking-out " threshold.

It has been demonstrated that, when the pilot is merely " blacked out " at his threshold value, general muscular control is relatively unimpaired, co-ordinated movements are still possible, and the oculomotor apparatus is apparently unaffected. This must indicate, therefore, that there is no major disturbance of the motor, pre-motor, or frontal cortex. General sensation would appear to be intact, although detailed analysis by clinical methods was impossible to carry out. It can also be assumed that there is no marked impairment of the function of the auditory or olfactory cortex. As regards the integrity of the occipital cortex, the voluntary ocular movements did appear to be supported by the fixation reflex in that the eyes could be kept accurately to one side or could be rigidly located on the position of the test card with strong rotation of the head. If the fixation reflex be maintained then there may be no gross impairment of the visual cortex (Holmes, 1938). However, the evidence cannot be taken as conclusive since the duration of the rise in retinal threshold was no more than 12–15 seconds at most. The occurrence of esophoria or of ptosis can be ascribed to latent muscular weakness or imbalance, rather than to paresis.

If the value of centrifugal force be markedly above the individual's threshold, impairment or total loss of consciousness may ensue. The unconsciousness, presumably, is due to cerebral anoxæmia, but disturbance in ionic balance, and fall in glucose content of the cerebral blood vessels may also be partly responsible. If the unconsciousness be due primarily to anoxæmia, one might expect to be able to study selective effects on the general musculature of deprivation of the higher nervous centres of the requisite oxygen. Those neurones which require the greatest oxygen quotient and the greatest vascularity, might be primarily affected. In every instance, however, the primary effect appeared to be loss of posture which occurred before complete paresis of the upper limbs. As has been pointed out above, this effect may be due to general loss of muscular tone, and the apparent primary location in the trunk musculature may be due to the relative increase in weight under G. Return of consciousness is remarkably rapid and this may not be without physiological significance. As in " blacking out " where return of vision is also dramatically swift, and where complete visual loss is rarely succeeded by a period of veiling, this may indicate that there is no absolute deprivation of blood from retinal or cerebral vessels.

It is obvious that great use can be made of the application of centrifugal force as a means for investigating, not only cortical function in man, but also its dependence upon the biochemical properties of blood and the physical interrelationships of C.S.F., venous and arterial pressures. Likewise a new field of visual investigation is opened up. It is known that the intra-ocular pressure is largely dependent on the general systemic arterial pressure, but that there are normally present mechanisms which tend to modify, or control, the effect of fluctuations in the systemic pressure. The use of centrifugal force presents new opportunities for investigation of this problem.

It has been noted that clonic twitching of the limbs has been observed both in these series of tests and in Diringshofen's series (1941). The frequency of fits in association with unconsciousness under G cannot be given, owing to the small number of cases (50), and in these complete unconsciousness was only photographed on eleven occasions. However, these clonic fits when present were associated with deep unconsciousness, but not with any abnormalities in the resting encephalogram or with any previous history of fainting.

Lastly, it has been observed that, although there is no gross impairment of cortical function at the " blacking-out " threshold, repeated " blacking out ", or exposure to low values of G, does lead to subjective fatigue. The physiological basis of this fatigue is unknown, but it is believed that it may result in part from minor degrees of cortical anoxæmia. There is insufficient increase in oxygen consumption to account for the fatigue on a physical basis, and it is similar to the fatigue which follows collapse on a tilting table. Thus, application of centrifugal force may also be valuable in the study of physiological fatigue.

Summary

(1) Some observations are presented which support the view that there is minimal impairment of cortical function at the " black-out " threshold.

(2) Application of centrifugal force as a method of experimental investigation holds marked possibilities for the study of fatigue, physiology of vision, of cortical function as well as circulatory problems.

It is a pleasure to thank Air Marshal Sir H. E. Whittingham for permission to publish this article, and Dr. B. H. C. Matthews, F.R.S., for much help and guidance in the research.

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NIGHT VISUAL CAPACITY OF NEUROTIC SOLDIERS

BY

W. LINFORD REES

(RECEIVED 22ND MARCH, 1945)

NIGHT blindness has been known since the time of the ancient Egyptians and was described by Hippocrates in the second book of Prorrhetics. One thousand years after Hippocrates, the beneficial effects of liver on night blindness were described by the Byzantine physician Paulus Aegineta. Rhazes recognized no less than three varieties (Holcomb, 1934). Richerand described an unrecognized occupational neurosis in the eighteenth century marked by night blindness and photophobia, caused by exposure to intense illumination (Culpin, 1940). The study of night vision, though always of interest, is of greater practical importance in war time, in view of black-out conditions, night driving, and night operations.

The ability to see in diminished illumination shows considerable individual variation. Minor degrees of night blindness are common. Maitra and Harris (1937) found that of 200 poor-class children, 20–30 per cent. were slightly below normal and 20–30 per cent. definitely subnormal in dark adaptation. Bishop Harman (1941) found that 14–28 per cent. of the normal population see poorly at night, the percentage increasing with age. Rycroft (1942) reported that 7–14 per cent. of four regiments tested were night vision defective. Lister and Bishop (1943) found that 3·9 per cent. of 10,333 first-class soldiers had poor scotopic vision.

Night blindness may be due to pathological eye changes, congenital and hereditary causes, vitamin A deficiency, pathological changes in liver, or over-exposure to light, Duke Elder (1938). Deficiency of vitamin A is the most widely accepted cause for defective night vision, and because of this it is estimated by dark adaptation tests, Harris and Abbasy (1939). Jeans and Zentmire (1934) produced normal dark adaptation by the administration of vitamin A in the majority of a group of children suffering from defective dark adaptation, and similar results were also obtained by Schuck and Miller (1938).

Some cases of night blindness have no apparent pathological or nutritional causes, and a number of writers have described hysterical or neurotic types. H. Smith (1921) regarded night blindness as malingering and postponed the publication of his paper until the end of the War, so as not to aid the enemy, who were discharging large numbers of men for the condition. Derby (1921) described "epidemics" of night blindness in the last war; Duke Elder (1938) pointed out that most patients without pathological changes or nutritional disturbances have neurasthenic and other functional symptoms also, while Livingston and Bolton (1943) suggest that some of the variations in night visual capacity recorded in healthy people may depend on differences in mental make-up. Wittkower, Rodger, *et al.* (1941) in a study of 52 soldiers complaining of night blindness found that the majority were suffering from severe psychological disorders. Over a half gave a history of over-dependence, neurotic traits, morbid fears, and excessive fear of darkness; while fears of injury (especially to the eye) were commonly seen and long antedated the onset of the disorder. Livingston and Bolton (1943), in an investigation of 50 patients suffering from neurosis, found that those with anxiety states performed the night visual capacity test poorly, whereas depressives and obsessionals gave satisfactory scores.

The purpose of this present study is primarily to determine whether a random sample of soldiers suffering from neurosis differed significantly in night visual capacity from normal control groups; secondarily whether the various neurotic types differed significantly from each other; and finally to find what characteristics or traits, if any, are associated with poor and good night vision.

The Material

The experimental population consisted of 96 soldiers (other ranks) suffering from clearly defined neurotic syndromes successively admitted to Mill Hill Emergency Hospital. Cases with mixed types of neurosis were rejected in order to facilitate the interpretation of the results. They were unselected regarding night visual capacity, and the psychiatric diagnosis was made by trained psychiatrists before testing, so that the subjective factor in their selection was reduced to a minimum.

The total group consisted of 36 men suffering from anxiety states, 33 men with depressive states, and 27 hysterics. They were all on full hospital diet, and none suffered from any marked visual defects.

The Technique

The men were tested by means of the Livingston Rotating Hexagon, an apparatus primarily designed for testing night visual capacity of R.A.F. personnel. The test was carried out by a W.A.A.F. Officer who was a trained Orthoptist and experienced in the use of the

apparatus. The test procedure was exactly the same as that used in testing R.A.F. personnel. A detailed description of the use of the Hexagon is given by Livingston (1942). Described briefly, the hexagon, which can be rotated so as to present different panels to the subject tested, has 96 letters and objects distributed on its 6 sides. The letters are placed in various positions, and the objects are in forms of aircraft, ships, parallel lines, etc. Preparation for the test includes 30 minutes dark adaptation, with dark goggles, only admitting 3 per cent. light, followed by 10 minutes in the dark room during which the details of the test are carefully explained. That this is an adequate period of dark adaptation is confirmed by the experiments of Yudkin (1943) who found that though 15 minutes, the time used in some tests, was inadequate, there was no improvement in dark adaptation after 35 minutes with the various types of apparatus tested. The subject is able to record his interpretation of the objects and letters in the dark by means of special Braille cards.

Four routine tests are given at different levels of illumination, each concerned with 6 letters and 2 objects, giving a total of 32. In each test a minute is allowed to record the answers with due warning after 45 seconds. A fifth test can also be given to detect any possible malingerers, or patients with hysterical amblyopia. In this, 3 large capital letters are exposed against a background sufficiently illuminated to ensure that they would be easily discernible except by those suffering from advanced pathological conditions, (Livingston and Bolton 1943).

During the explanation of the test the patients were encouraged to do as well as possible and to compete with each other, and told that they would be able to see the objects more clearly if they gazed a little to the side of the object. In order to allay any suspicions about the test, it was carefully pointed out that it was a scientific investigation, and was not in any way relevant to their treatment or disposal.

Lythgoe (1940) stresses the dual mechanism of dark adaptation. The first part of the process lasts 6–11 minutes and is concerned with the adaptation of the photopic mechanism of vision. The second phase is the adaptation of the scotopic mechanism mediated by the retinal rods and their visual purple. Visual sensitivity during dark adaptation may increase 10,000 times.

The Livingston Rotating Hexagon test examines a mixture of rod and cone vision, the background brightness during the test ranging from 0·00015 eq. ft. candles to 0·0012 eq. ft. candles. Each apparatus is calibrated by a skilled technician in photometry to ensure that the light values of each apparatus are comparable.

Results

The results of the total group of 96 men are shown in the frequency histogram, Fig. 1. Fig. 2 shows the results of night visual capacity testing of 6,062 R.A.F. personnel with the Rotating Hexagon,

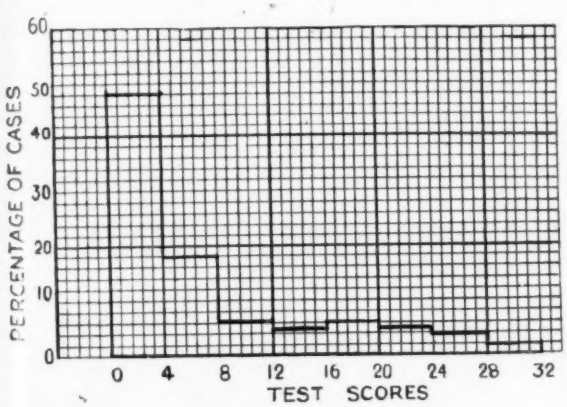

FIG. 1.

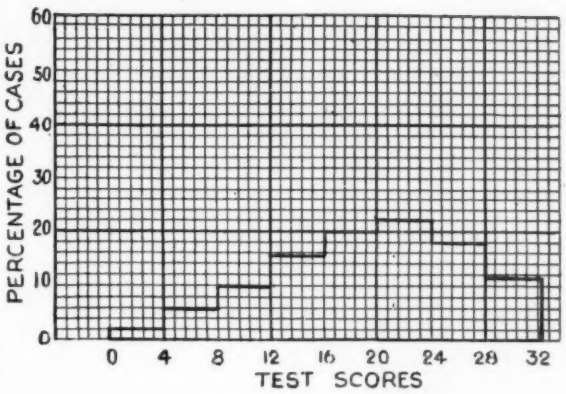

FIG. 2.

which were kindly supplied by Air Commodore Livingston.

It will be seen that whereas the results of the R.A.F. personnel group are distributed in a normal frequency or Gaussian curve, the frequency distribution of the neurotic group is in the form of a reversed " J "-shaped curve.

Inspection of the two frequency distributions indicates that the performance of the neurotic group on the test is markedly inferior to that of the R.A.F. group. This impression is confirmed by statistical comparison of the means. The mean night visual capacity of the R.A.F. group is 19·32, standard deviation 3·5, that of the neurotic group is 7·1, with a standard deviation of 8·05. The difference between the means is highly significant statistically (critical ratio=14·84). The Median of the neurotic group is 4·0 and the values of the 25th and 76th percentiles 1·92 and 12·0 respectively. The corresponding figures for the R.A.F. group are 18·99, 14·48, and 24·8. In view of the dissimilarity between the two frequency distributions the chi square test was also applied. X^2 was found to be 984·48 (P=considerably less than 0·01), which again indicates that the difference between the two groups is of high statistical significance.

We conclude, therefore, that the performance of the neurotic group on the test is significantly inferior to that of the R.A.F. group.

Neurotic types

Table I shows the results obtained by the various types of neurosis.

TABLE 1.

Type	No. of cases	Test score				Mean	Median
		0–7	8–15	16–23	24–32		
Anxiety state	36	29	4	3	0	4·8	3·2
Depressive state ..	33	22	3	4	4	7·9	3·5
Hysteria ..	27	17	4	4	2	8·7	5·2

Anxiety State Group.—Eleven men of this group complained that they were unable to make out any letters or objects though some had obtained a few marks. These men were tested with the top panel with bright illumination in order to discover malingerers or hysterics, one man only gave a result suggestive of malingering or hysteria. The group is significantly inferior in performance on the test to the R.A.F. group. ($X^2=512{\cdot}6$, P is considerably less than 0·01.)

Depressive State Group.—Five of this group of 33 subjects complained that they were unable to see anything. They were tested with the top panel but none gave the hysterical or malingering response. The results are somewhat better than those of the Anxiety State Group but the difference is not statistically significant ($X^2=1{\cdot}42$. $P=0{\cdot}99$). The performance of the group is significantly inferior to that of the R.A.F. Group. ($X^2=177{\cdot}9$. P is less than 0·01.)

Hysteria Group.—Eight of the group of 27 hysterics complained that they were unable to make out the objects and letters. These men were accordingly tested with the top panel, but none gave the malingering or hysterical result. The performance of the group in the test is somewhat better than that of the Anxiety State and Depressive State Groups, but the differences are not statistically significant. (Hysteria and Anxiety State $X^2=8{\cdot}36$. P is between 0·2 and 0·31. Hysteria and Depressive State, $X^2=1{\cdot}69$. $P=0{\cdot}97$.)

Comparison with other Normal Groups

The group of 6,062 R.A.F. personnel is not ideal as a control group, as the standards for enlistment in the R.A.F. are more rigorous than for the Army, so that the R.A.F. results may be higher than those of the general population of the British Isles. Unfortunately data for results of the test on the general population are not available. Steadman (1942) tested men of an Anti-Aircraft Battery with the Livingston Rotating Hexagon, and the percentage results obtained are given in Table II, together with data on 2,000 R.A.F. personnel. Steadman graded the results as follows:—

Exceptional	29–32	Below average ..	3– 8
Above average	20–28	Poor	0– 2
Average ..	9–19		

The neurotic groups were graded similarly and the percentage distribution is shown in Table II.

The results shown in the table confirm our previous conclusions and show in a striking manner the weighting of the total neurotic group, and component neurotic groups towards the lower end of the scale of night visual capacity.

Age Factor.—That night vision becomes worse with increasing age has been shown by Ferree *et al.* (1935 and 1938), Bishop Harman (1941), Rycroft (1942), and Lister and Bishop (1943). It is therefore important to determine whether the age factor has a significant influence on our results. The mean age of the total group is 30·2 years, standard deviation 6·6. The product moment correlation between age and night visual capacity test score for the total group was found to be $-0{\cdot}394 \pm 0{\cdot}086$. This negative correlation is statistically significant and indicates that the older age groups tend to have poorer night visual capacity. The mean age of those performing well on the test (19 and over) is 27·6 years, standard deviation 6·04, and that of the group performing very poorly (4 and under) was 32·2 years, standard deviation 7·04. The difference of low statistical significance (critical ratio=2). Thus there is a definite association between age and score on the test. Lister and Bishop (1943), however, found that using the Army night vision test on 10,333 soldiers, there was a steady fall in night visual capacity from the age of 18, but that the fall between 18 and 44 was so slight as to be of little practical importance.

Intelligence Factor

Rycroft (1942) states that intelligence plays a part in the results of night vision test results, the more intelligent tending to do better. In order to test the possible influence of intelligence on the results, each man was given Raven's Progressive Matrices Group Intelligence Test, and the results were graded in the usual way into five grades, grade 1 indicating superior intelligence and grade 5 poor intelligence. The Pearson product moment correlation coefficient was calculated for night visual capacity score and Matrices grade, and was found to be $r=0{\cdot}107 \pm 0{\cdot}1$. The correlation is only slightly greater than its standard error, and is not statistically significant. The Mean Matrices grade for the total group was 2·69 (standard deviation 1·16). As Grade 3 is the accepted grade of average intelligence, the mean grade of the total neurotic group is somewhat above average intelligence and, therefore,

TABLE 2.

Grade	A.A. men	Officers	R.A.F.	Anxiety state	Depressive state	Hysteria	A total neurotic group
Exceptional	1·4	20	3·5	0	3·30	3·703	2·83
Above average	18·2	26·7	32·8	2·77	12·12	14·81	9·375
Average	49·1	53·3	49·6	16·66	18·18	18·51	17·17
Below average	23·1	0	13·1	30·55	24·24	25·92	28·12
Poor	8·2	0	1·0	50·0	42·42	37·03	43·75
Average marks	12·9	19·6	—	4·8	7·9	8·7	7·105
Number	151	15	2,000	36	33	27	96

we are unable to explain the poor results of the total group on the intelligence factor.

As the extreme ranges of intelligence may have cancelled each other out within the groups, it was decided to compare the intelligence grades of those who had scored poorly (4 and less) with those who had scored well (19 and over).

The results are given in Table III.

From the table it will be seen that in the good night vision group the percentage increases in the "above average" intelligence grades (Grades II and I), whereas, in the poor night vision group, the proportions are normally distributed. Though there are considerable differences in the percentages in the grades indicating a higher intelligence in the good night vision group, the differences are not statistically significant.

In the good night vision group 76·9 per cent. are above average intelligence, whereas, in the poor night vision group only 30 per cent. are above average intelligence. The difference is statistically significant (critical ratio=2·76). Thirty per cent. are below average in poor night vision group, whereas only 7·69 per cent. of the good night vision group are below average intelligence. The difference is statistically significant (critical ratio=2·21). Thus the group with good night vision has a demonstrably higher intelligence as measured by the Matrix test.

Effect of Suggestion on the Test Results

Nine men who had obtained low scores (2 and under) were given a talk in which they were firmly told that better results were expected of them, and strong suggestion was given to the effect that they would improve with retesting.

Seven patients had Anxiety states; of these, one increased his score from 0 to 1, and another from 1 to 4; 3 men obtained the same scores, and 2 decreased their scores from 1 to 0, and 2 to 1. The remaining 2 patients had Depressive states; one patient decreased his score from 3 to 1, and the other obtained the same score as before. Three men improved their scores, gaining a total of 4 points, and 3 men performed more poorly, losing a total of 4 points, thus, the resultant effect of suggestion was nil. In no case did sufficient improvement occur to promote the patient from the very poor group (scores of 4 and under).

Comparison of Good Night Vision and Poor Night Vision Groups.—In order to determine whether good and poor night vision are associated with characteristic traits, a group of 50 men with poor scores (4 and under) were compared with the group of men (13) who had scored well (19 and over), in respect of over 150 items which had been scored by the physician in charge of the case on a special item sheet before the night vision test was carried out. The percentage distribution of the various items in the two groups was calculated and the significance of the difference determined by means of the Critical Ratio (C.R.).

The following is a brief account of the results.

Social Data.—The proportion of N.C.Os. is higher in the good night vision group (30·7 per cent. compared with 16 per cent. in poor group). There were no significant differences in type of service occupation, duration of service, modal civilian occupation and pre-service earnings. Twenty-eight per cent. of the poor group had considerable unemployment compared with nil in good group (C.R. =4·41). Similarly the work history showed unduly frequent changes of occupation in a significantly greater proportion of the poor group (10 per cent. poor group; C.R.=2·37). An indication that the men with poor night vision are on the whole poorer material is given by the fact that 46 per cent. were considered to be of no further use to the Army, and were discharged category "E." This proportion is statistically significantly greater than that of the group with good night vision (15·38 per cent., C.R.=2·43).

Family History.—There was a higher incidence of a family history of neuropathy in the poor group, but the differences were not statistically significant.

Personal History.—Most of the items relating to the personal history revealed a greater incidence of unsatisfactory factors in the poor night vision group. Thirty per cent. of the poor group did not pass standard five at school, and this is a significantly higher percentage than that found in the good night vision (7·69 per cent. C.R.=2·29). The group with poor night vision had a much smaller proportion of individuals taking active interest in social groups, and 84 per cent. were described as having narrow interests. Twelve per cent. of the poor group had a history of excessive alcohol consumption, whereas none of the good group had such a

TABLE 3.

Intelligence grade	Poor night vision group N=50 (per cent.)	Good night vision group N=13 (per cent.)	Difference in percentage	Standard error of difference	Critical ratio
V	8	7·69	0·31	8·59	0·03
IV	22	—	22·00	5·55	3·75
III	40	15·38	24·62	12·50	1·96
II	22	46·15	24·15	15·53	1·55
I	8	30·75	22·76	13·86	1·64
Mean grade ..	3·0	2·07			

history (C.R.=2·61). The percentage of the poor night vision group having normal mental health before the present illness was only 16 per cent. compared with 61·53 per cent. of the good night vision group; the difference is statistically significant (C.R.=3·04). Sixty per cent. of the poor group had a clear predisposition to neurotic illness compared with 23 per cent. of the good night vision group (C.R.=2·6). Similarly, a statistically significantly higher proportion had previously had a definite mental illness (16 per cent. and nil respectively) (C.R.=3·08). Thus on the whole the personal history of the group with poor night vision is less satisfactory than that of the group with good night vision.

Personality.—Only 30 per cent. of the poor night vision group had a well-organized stable personality compared with 76·92 per cent. of the good group; the difference is statistically significant (C.R.=3·4). Similarly, the percentage with weak, dependent, timorous personalities is significantly greater in the poor group (66 per cent. compared with 23·08 per cent. C.R.=3·0). Cyclothymic tendencies also occurred in significantly greater frequency in the poor group (52 per cent. compared with 7·69 per cent. C.R.=3·6). Similarly, 22 per cent. of the poor group were regarded as being very anxious and highly strung, whereas none of the good group fell into this category (C.R.=3·7). Obsessional traits occurred in 14 per cent. of the poor night vision group, and in none of the good group (C.R.=2·85). There were no significant differences in the proportion of schizoid, hysterical or hypochondriacal traits.

Symptoms and Findings.—In a comparison of 30 items, the following showed statistically significant differences:—

Suicidal tendencies (16 per cent. in poor group, none in good group : C.R.=3·08). Stammer (8 per cent. poor group, nil in good group : C.R. =2·08). Poor muscular tone and posture (10 per cent. poor group, nil in good group: C.R.=2·3).

Discussion

Our results show that the neurotic group performed more poorly on night vision testing than the various normal groups considered. On subdividing the group into diagnostic types it was found that they did not differ significantly from each other on the test but were significantly inferior to the R.A.F. normal group. Within the total neurotic sample we find that the group with low scores differs significantly from the group with high scores in a number of psychiatric traits.

The group of individuals with low night vision scores is associated with a clinically abnormal personality (as shown by general instability, weakness and dependence, cyclothymic tendencies, anxious and highly strung, obsessional traits and narrow interests) a positive family history of neuropathy, previous mental illness or clear disposition to neurosis, or a poor school and work record.

These findings are of particular interest in view of recent work by Slater (1943), who found that the above traits were the best indicators of the neurotic constitution, thus showing that the group of individuals performing poorly on the night vision test are of more marked neurotic constitution than those who did well.

The reason for the poor performance on the night vision test of the markedly neurotic individual is not clear. The possibility that the poor results are due to malingering can be ruled out as a special test for malingering was applied, and only one individual gave a result indicative of malingering. It seems possible that the poor performance of the neurotic group on the test might be due to a defect at the perceptual level and that the neurotic, through possible lack of incentive or other cause would tend to do poorly on a complex perceptual test of this kind. Although an attempt was made to counteract any lack of incentive, it is not completely possible to rule it out as a possible factor. It is, however, difficult to see why lack of incentive should operate in this test and not in the matrices intelligence test—a complex perceptual test in which this group performed better than the average.

Kekcheyev *et al.* (1943) point out that sensitivity of night vision is one of the most fluctuating functions of the human organism and that such factors as fatigue, distension of visceral organs, and strong stimulation of other sense organs may diminish night vision. The same authors found that the effect of conditioned reflexes on night visual acuity may be strong, so that poor night vision may result from any fears associated with darkness. In order to test this possibility the medical records were examined, and it was found that 60 per cent. of the group of men with poor night vision had marked fears of the dark, either in childhood or adult life, whereas only 27·7 per cent. of the good night vision group had similar fears. The difference is statistically significant (C.R.=2·3). Thus fear of the dark may in some cases be a factor in the pathogenesis of poor night vision.

Although a number of features such as age, intelligence, neurotic constitution, and certain psychiatric traits were found which show a definite relationship with performance on the test, the precise nature of the defect in the neurotic group has yet to be elucidated.

It is possible that the results do not indicate a specific defect, but a general deficiency which predisposes to the development of neurosis. It would seem that the results support a recent heuristic theory of neurosis postulated by Slater and Slater (1944). This heuristic theory states that the neurotic constitution is predominantly determined by a large number of genes of small effect. The effects of these genes, at least in so far as they are qualitatively similar, may be additive and they become manifest by producing a reduced resistance to some form of environmental stress, and so facilitate the appearance of neurosis. The authors found that neurotics, on the average, had poorer auditory and day-time visual activity than normal control groups.

A deficiency in such abilities together with other attributes of the neurotic constitution would tend to lower resistance to stress and facilitate the development of neurosis.

Summary

1. A random sample of 96 neurotic soldiers were tested with the Livingston Rotation Hexagon and were found to be considerably below the normal population in night visual capacity.

2. Dividing the group into neurotic types it was found that they did not differ significantly from each other but were significantly inferior in night vision test score to the R.A.F. normal group.

3. Age and night vision test score showed a significant correlation, older individuals tending to score more poorly on the test.

4. The correlation between intelligence grade and night vision score was insignificant for the group as a whole. The group with good vision scores, however, had significantly greater proportion of individuals above average intelligence than the group with low night vision scores.

5. Suggestion was found to have no significant effect on the score obtained.

6. The group with poor night vision scores was compared with a group obtaining good scores. The poor night vision group had a lower standard of education, poorer work history, narrower interests, greater incidence of alcoholism and previous mental illness, a greater predisposition to neurosis, a higher proportion of weak, dependent, anxious, cyclothymic and obsessional types of personality and a greater incidence of suicidal tendency, stammer and poor muscular tone and posture. There was also a greater incidence of fear of the dark either in childhood or adult life among individuals with poor night vision scores. In short, the group of individuals with poor night vision scores presented a more marked neurotic picture with greater constitutional predisposition to neurosis.

7. The findings are discussed in relation to a recent heuristic theory of neurosis.

I wish to thank Dr. W. S. Maclay for permission to publish the case material and Air Commodore Livingston for valuable advice.

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LESIONS OF THE CENTRAL NERVOUS SYSTEM IN HERPES ZOSTER

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(RECEIVED 2ND MAY, 1945)

SINCE Head and Campbell's classical description of the pathology of herpes zoster, it has been recognized that the principal sites of damage in this disease are the cell bodies of the first sensory neurones in the posterior root ganglia and the ganglia associated with certain of the cranial nerves. More recently, however, it has been emphasized, especially by Lhermitte, that this is not the only lesion possible, and that the virus can attack the central nervous system as well. Herpes zoster, like other virus diseases of the nervous system, may thus be considered as an infection involving the nervous system as a whole, though having a predilection for one particular type of nerve cell. Three cases seen recently seem to illustrate this aspect of herpes zoster and may therefore be of interest.

Cases in which the central nervous system has been affected, normally fall into one of the following groups; firstly, those in which the anterior horns of the spinal cord are affected (anterior poliomyelitis); secondly, those which show evidence of involvement of the pyramidal or other tracts of the white matter of the spinal cord (transverse myelitis); thirdly, those in which the brain is involved (encephalitis). Individual cases may sometimes fall into more than one of these groups.

The three cases seen personally appear to be examples of each of the three groups, the first showing involvement of the lower motor neurones of the second and third lumbar segments following a zoster of the corresponding dermatome; in the second case a paraplegia with sphincter disturbances followed herpes of the 10th and 11th dorsal segments; in the third case a left ophthalmic herpes was followed by a right hemiplegia, aphasia, and pupillary abnormalities.

Case 1.—A.W. Male, aged 68.

History of present illness: He was admitted to hospital complaining of weakness and numbness of the left leg and the left side of the face, which came on in the leg over a period of two to three days about five weeks previously, and seems to have spread from the foot upwards to include the lower part of the abdomen. At the same time as the onset of the weakness and numbness, there appeared a crop of small blisters over the whole of the inner side of the left leg, from below the knee to the groin. These lasted about a week, leaving a thin papery scar. While the rash was present there was a fairly severe "drawing" pain involving the whole of the left leg. There was no pyrexia, but he slept badly and felt depressed and muzzy in the head.

Two weeks later, he noticed that he could not shut his left eye and that his mouth was crooked. He also complained of a feeling of constriction round his trunk at the level of the iliac crests. He vomited on one occasion but did not complain of headache or disturbance of eyesight or hearing. His appetite was poor and his bowels constipated.

Previous history: Had been abroad for forty years, where he contracted various types of fever. Nothing else significant.

Family history: Nothing significant.

Examination on 22.10.41 showed a healthy-looking man with normal temperature, pulse rate 82, regular and of good volume; B.P. 135/95; with no abnormalities in the cardiovascular, respiratory, and digestive systems.

C.N.S.: The fundi were normal; visual acuity and fields normal; pupils equal, regular, reacting to light and accommodation; full eye movements with no nystagmus; slight weakness of movements of the left side of the face; facial sensation and corneal reflexes normal; the other cranial nerves were normal.

In the upper limbs there was no abnormality in muscular power, tone, or coordination, and no tremor. Sensation was normal and the tendon reflexes brisk and equal.

In the trunk, muscular power was normal; touch and pain sensation were slightly diminished over the lower part of the abdomen on both sides. The abdominal reflexes were present on the right but absent on the left side.

In the lower limbs, there was considerable loss of power, most marked in the flexors and adductors of the left hip and in the left quadriceps and hamstrings, less marked in the abductors and rotators of the left hip and the anterior tibials. The glutæi and posterior tibials were apparently unaffected, the tone of the weak muscles was reduced. There was a small area of complete loss of touch, pain and temperature sensation on the inner side of the left knee, and the upper third of the inner side of the shin, with a surrounding area of diminished sensation over the front and inner side of the thigh, and the inner side of the leg down to the internal malleolus on the left side. Vibration and postural sense were unaffected. The left knee jerk was absent, but the left ankle jerk was present and about equal to that on the right side. The plantar reflexes were both flexor.

A lumbar puncture was performed on 25.10.41, and a faintly yellowish fluid was obtained under normal pressure. Other findings were:

Cells: 3 lymphocytes/cu. mm.
Protein: 120 mgms. per cent. Globulin positive (Nonne).
W.R.: Negative.

The W.R. in the blood was also negative.
The abnormal findings were therefore as follows:—
Slight L. sided facial weakness.
Weakness and hypotonia of both legs.
Area of anæsthesia and analgesia in the L. leg.
Absent L. abdominal reflexes.
Absent L. knee jerk.
Xanthochromia and increased protein in the C.S.F.

Progress: During his stay in hospital there was considerable improvement in the weakness and numbness, but he complained of severe neuralgic pains in the left leg necessitating the use of sedatives.

After his discharge home, the weakness of his left leg became much worse again, and he was unable to get about for the next fourteen months. Improvement, however, gradually occurred with massage and active movements, and when seen on 24.6.43 he was able to walk with the help of crutches. He complained, however, of pins and needles in both hands and of a moderate amount of pain in the left leg. His feet were always cold. Otherwise his health was good.

Examination at this time showed normal fundi; pupils equal and regular, reacting briskly to light and on accommodation; full eye movements with lateral nystagmus; very slight facial asymmetry, but no weakness of the facial muscles was detected; other cranial nerves normal.

No abnormality in power, tone, coordination, sensation or reflexes was found in the upper limbs.

In the trunk there was tenderness on pressure in the 9th intercostal space at the back, but otherwise no abnormality of power or sensation; the abdominal reflexes were all present and equal.

In the lower limbs there was still demonstrable weakness in the left quadriceps, hamstrings, and the rotators of the left hip. The power of the other muscles of the left leg and of the muscles of the right leg was normal. Tone and coordination of all muscles appeared normal. Sensation was normal except for slight diminution of vibration sense in the left leg. The knee and ankle jerks on the left side were both absent even with reinforcement. The plantar reflexes were both flexor. The gait was abnormal; a definite limp was present, though there was no ataxia or Rombergism.

Case 2.— T.P. Male, aged 47. Occupation, fitter.

History of present illness: Eight weeks before admission to hospital he suffered from a typical attack of herpes zoster affecting the skin of the trunk at the level of the 10th and 11th dorsal segments.

Four days prior to admission, there was a sudden onset of acute stabbing pain in both loins radiating round to the front of the abdomen. Some hours afterwards his left leg became numb and he was conscious of paræsthesiæ. When he stood up he found he was unable either to walk or stand normally, and his left leg was paralysed. The right leg was weak but not so severely affected. He was also unable to pass his water.

On the day of admission he passed small amounts of urine frequently. The right leg had recovered almost completely and the left leg was recovering some movement.

He had had a transient attack of blurring of vision, but otherwise his previous health had been good with no urinary or dyspeptic symptoms. He denied exposure to syphilis.

Examination on 25.5.42 showed a healthy-looking man with no abnormalities in the respiratory or cardiovascular systems; the bladder could be felt as high as the umbilicus.

C.N.S.: The fundi showed a doubtful temporal pallor of the right disc; there was coarse lateral nystagmus; speech was normal; the other cranial nerves were normal.

In the upper limbs power and tone were normal; tendon reflexes were present and equal; there was slight intention tremor; sensation was normal.

In the trunk the abdominal reflexes were all absent; there were scattered areas of loss of perception to light touch below the umbilicus; pain and deep pressure sense were normal.

In the lower limbs, the right leg showed normal muscular power and tone; the knee and ankle jerks were present; there was no clonus; the right plantar reflex and Oppenheim were extensor; there was no Strumpel and no abnormality of any form of sensation. In the left leg muscular power was almost completely absent; tone was increased with a "cog-wheel" rigidity; the knee and ankle jerks were both exaggerated as compared with the right side; there was no clonus; the plantar reflex and Oppenheim were both extensor, with a positive Strumpel; all forms of sensation were impaired; no muscle wasting was observed in either leg.

A lumbar puncture was carried out and 10 c.c. of clear colourless fluid was obtained under normal pressure. Other findings were:—
Cells: 100 red cells; no white cells.
Protein: 35 mgms. per cent.; Globulin: negative.
Lange: 1233400000.
W.R.: negative.

In brief, the abnormal findings were as follows:—
Doubtful temporal pallor of R. disc.
Nystagmus.
Intention tremor.
Weakness and spasticity of L. leg.
Diminished sensation over abdomen and L. leg.
Absent abdominal reflexes.
Increased knee and ankle jerks on L. side.
Plantar reflex and Oppenheim extensor both sides.
Retention of urine.
Abnormal Lange curve in C.S.F.

Progress: At first the patient required repeated catheterization. Following discharge from hospital he improved slowly, and finally was able to get about pretty well and walk most of the day without getting tired. There was, however, always some weakness of both legs as compared with his condition previous to admission. The sensation of constriction round the waist persisted and he had had urgency and frequency of micturition. He had only had proper control over his bowels for the last six months. He was now constipated.

For the last six months the weakness and stiffness in the legs had been getting worse again. He got tired easily and could only walk about a mile. The frequency and urgency of micturition had also been worse, as was the pain. His appetite was good and his weight steady. He slept well; there were no flexor spasms or pain at night, though he was disturbed by the frequency. He had had no further trouble with his eyesight.

Examination on 18.10.44 showed a healthy-looking man; the fundi showed deep physiological cups but were otherwise quite normal; the pupils were moderately dilated, reacted briskly to light and on accommodation; ocular movements full, with no nystagmus; the other cranial nerves were normal.

In the upper limbs, power tone and coordination were normal, reflexes were normal, and there were no sensory abnormalities.

In the trunk the abdominal reflexes were not brisk, but were present and equal; muscular power was normal; there were vaguely defined areas of hypoæsthesia and hypalgesia over the abdomen.

In the lower limbs, there was slight weakness in flexion of the left knee; power was otherwise normal; there was moderate spasticity in both legs; coordination was somewhat impaired. There was gross ataxia and he walked with a wide base; there was slight Rombergism. Sensation was normal apart from some diminution of vibration sense; the tendon jerks were brisk and equal; the plantar reflexes were still both extensor; no Strumpel was present.

Case 3.—H.A. Male, aged 26. Private soldier.

History of present illness: About the middle of July 1944 he was admitted to the 30th Gen. Hospital with a left-sided herpes ophthalmicus and œdema of the left eyelid. He complained of general malaise and left-sided frontal headache which he had had for five days.

On examination at this time there was a herpetic rash over the distribution of the ophthalmic division of the fifth nerve and right submaxillary adenitis but no corneal ulceration. After his evacuation to this country slight ciliary injection developed in the left eye.

Three weeks later he suddenly felt queer and complained of pain in his right arm; there was no unconsciousness but he was unable to raise the arm. A partial paralysis of the right arm and the right side of the face was found on examination; speech was slurred. The paralysis was flaccid but with normal tendon reflexes, no wasting and no sensory abnormalities; it did not involve the right leg or the left side of the body; the plantar reflexes were flexor and the abdominal reflexes present. A lumbar puncture done at this time showed xanthochromia, 3 cells per cu. mm., normal protein and chlorides and a negative W.R.

A week later the paralysis of the right arm became more pronounced and the right leg began to be affected; the pupils became unequal, the left being larger than the right but both reacting to light and on accommodation; speech became gradually more and more difficult, developing into a gross aphasia.

The herpes had almost cleared before the onset of the paralysis.

Previous history and family history yielded nothing significant.

Examination on 3.9.44 showed a well-developed man with no abnormality in heart, lungs, or abdomen. His temperature was 97, pulse rate 80, regular and of good volume; B.P. 130/84; urine normal.

A fairly gross degree of aphasia was still present, though it appeared to have improved slightly recently. The defect was predominantly expressive; he could form simple sentences but often got held up for lack of a word; he complained of difficulty in thinking of the right word to say, though he knew what he wanted to say and could recognize the word when it was said for him; he often used wrong words and pronounced them badly; reading was very slow and inaccurate; memory seemed fairly good, though he could not give a connected history; he could repeat words accurately and he could obey commands as long as they are not too complicated, but he had difficulty in distinguishing between left and right and in calculation. He had little nominal defect; he cooperated well and there was no confusion or disorientation. He stated that he had noticed that his speech became worse if he was tired or if he had been trying to read for a long time.

There was bilateral diminution in the sense of smell; a gross refractive error was present in the right eye and a slight one in the left eye; the discs were normal; the visual fields appear normal to rough tests; the pupils were equal and reacted briskly to light but only sluggishly on accommodation; eye movements were full with no ptosis or nystagmus; there was a right-sided upper motor neurone paralysis of the face; facial sensation was normal; the right trapezius was weak and the tongue was protruded to the right.

In the upper limbs, muscular power, tone, and coordination and all forms of sensation were normal in the left arm. There was weakness of all movements of the right arm; he could just flex the wrist, and could abduct the shoulder to a right angle, but the movement was definitely weak; muscle tone was increased; sensation was normal except for defective postural sense; there was no astereognosis present. The tendon reflexes were brisker on the right than on the left.

In the trunk, power and sensation were normal, but the right lower abdominal reflex was diminished.

In the lower limbs there was a just detectable weakness of movements at the right ankle and knee; tone was equal in both legs; there was some tremor in both legs but no clonus; posture and coordination were otherwise normal. There was no abnormality of sensation in either leg; the tendon reflexes were brisker on the right side than on the left; plantar reflexes were both extensor. The gait was of the typical hemiplegic type and there was some degree of pes cavus on both sides, more so on the right than on the left.

The following investigations were carried out:—

X-ray of skull: normal.

Lumbar punctures:

27.8.44.

Clear fluid; pressure, 140 mm.
Cells: 70 red cells; 12 white cells per cu. mm.
Protein: 60 mgm. per cent.
Sugar: 53 mgm. per cent.

4.9.44.

Clear fluid; pressure: 60 mm. with rapid rise and fall on jugular compression.
Cells: 2 polymorphs and 36 lymphocytes per cu. mm.
Protein: 70 mg. per cent. Globulin positive (Nonne.)
Lange: 1223320000.

Cisternal encephalography showed both lateral and the 3rd ventricle to be well filled and central in position. No evidence of a space occupying lesion was found.

In brief, the abnormal findings were:—

Aphasia.
Diminished sense of smell.
Pupils reacting sluggishly on accommodation.
R. hemiplegia with spasticity and increased tendon reflexes.
Bilateral extensor plantar reflexes.
Tremor of legs.
Diminution in postural sense in both arms.
Pleocytosis, positive globulin, increased total protein and abnormal Lange curve in the C.S.F.

Progress: The movements of the right elbow and shoulder improved rapidly, and when examined on his discharge from hospital on 21.9.44, there was marked general improvement. The aphasia, however, was still quite well marked, though he could talk sufficiently well to make himself understood. Fine movements of the right hand and fingers were still very poor, though movements at the right shoulder were fairly normal; leg movements were good and he could walk fairly well.

As none of these cases have died it has not been possible to investigate the pathological changes responsible for the clinical findings. It may be of interest, therefore, to review the findings of other authors in similar cases and the evidence they adduce in favour of lesions of the central nervous system in herpes zoster. They will be found to afford considerable support for the theory that the zoster virus can invade the central nervous system and to suggest a possible pathological basis for the clinical findings observed in the three cases just described.

Involvement of the anterior horn and the lower motor neurone in herpes is not uncommon. The site of the paralysis is usually segmental and related to that of the eruption. It is of the lower motor neurone type and comes on as a rule a few days after the appearance of the eruption and may persist for a period of years, though the ultimate prognosis according to Bing (1939) is always favourable. Cases of this type are described by Joffroy (1882), Waller (1885), Taylor (1895), Hardy (1876), Cornil (1930), Wilson (1940), and Carter and Dunlop (1941).

Cases showing involvement of the white matter of the spinal cord are rare. A monoplegia with or without sensory changes is the commonest clinical picture, but a complete paraplegia with sphincter disturbances, root pains, and complete sensory loss below the level of the rash has been described.

The symptoms of these cord lesions come on gradually as a rule from two to six weeks after the zoster and thereafter slowly improve, though signs of pyramidal involvement, such as weakness, extensor plantar reflexes, increased tendon reflexes which may be responsible for considerable disability, may persist for several years. The prognosis must therefore be regarded as uncertain.

In two cases of spinal cord symptoms following herpes zoster evidence of independent lesions have been discovered. This suggests that in certain cases the herpes occurs secondarily to an independent cord lesion which has been undetected until the herpes draws attention to it. The condition of "symptomatic herpes" is well known, and the whole question will be discussed more fully later on. The occurrence of this type of case is reviewed by Worster Drought and McMenemey (1934).

Evidence of damage to the brain following herpes zoster is also uncommon. The clinical picture in these cases is of two types, firstly a localized lesion very similar to the lesions in the spinal cord, usually resulting in a hemiplegia, and secondly a generalized encephalitis. Most of the cases of the first type have occurred in old age and have had a uniformly fatal outcome. Such cases are described by Fumery and Brissaud (quoted by Worster Drought and McMenemey 1934), by Lhermitte and Vermes (1930) and by Faure-Beaulieu and Lhermitte (1929).

The second type of case appears to be almost universally fatal. They have been described by Schiff and Brain (1930), Thalhimer (1924), Gundersen (1925), and Biggart and Fisher (1938). Netter (1930) has described cases in which the encephalitis preceded the zoster, but these are probably to be regarded, according to Schiff and Brain, as cases of "symptomatic herpes."

Most of these cases show evidence of meningitis as well, and indeed there is evidence that this occurs at some stage in the majority of cases of zoster, though it may be without clinical signs; at any rate a mild lymphocytosis and increase in protein in the C.S.F. is a common finding (Brain, 1940, Wiegmann, 1934).

As regards the pathological basis for these clinical findings the only changes in the central nervous system described by Head and Campbell (1900) were the degeneration of the posterior columns resulting from the destruction of the cell bodies in the posterior root ganglia, but they admit that herpes zoster is probably an acute specific disease of the nervous system as a whole with an initial febrile stage analogous to that of anterior poliomyelitis.

Lhermitte and his colleagues on the other hand have shown that even in uncomplicated cases, herpes zoster results not only in lesions of the posterior root ganglia, but also causes inflammation of the anterior horns of those segments of the cord in relation to the affected ganglia. Sections of the spinal cord show very well marked dilatation of the vessels, destruction of the nerve cells and fibres, neuroglial proliferation and scattered collections of lymphocytes; these changes are most marked in the region of the posterior horn of the grey matter, but also affect the base of the anterior horn, which shows loss of cell bodies and scattered lymphocytes. While they are most severe in the segment affected by the zoster, they extend to affect adjacent segments to a lesser degree (Lhermitte and Vermes 1930). Similar changes have been described more recently by Denny Brown, Adams and Fitzgerald (1944). These authors emphasize microglial proliferation with lymphocytic infiltration round the veins, which, though strictly unilateral, extends up and down the cord for two or more segments from that in which the eruption occurs. Damage to the nerve cells of the cord is slight but quite definite, and occurs particularly in the anterior horns, substantia gelatinosa, and Clarke's column. These authors also found, in two cases, degenerative changes in the anterior roots at the point at which they pass closest to the posterior root ganglia. They considered that these were far too extensive to be accounted for by the degenerative changes in the anterior horns and postulate that they are due to direct extension of the inflammatory process from the posterior root ganglion.

As regards damage to the white matter of the cord, Lhermitte and Nicolas (1924) found perivascular "cuffing" in the region of the pyramidal tract. Degenerative changes occur of course in the dorsal columns as a result of the damage to the cells of the posterior root ganglia.

Lhermitte and Vermes also describe the lesions they found in the brain stem and basal ganglia in cases of herpes zoster. In these the vessels are tortuous and distended with blood, there are scattered hæmorrhages, which, though small are numerous, and areas which appear translucent on section owing to the presence of "lacunæ," particularly in the region of the pyramidal tracts. In spite of the advanced age of most of the patients in which these lesions were found, these authors consider that they are directly due to the herpes virus on the grounds that they are of a similar type to the lesions in the spinal cord, and do not resemble the lesions due to arteriosclerosis or thrombosis.

Biggart and Fisher (1938) describe the pathological findings in their fatal case of encephalitis following herpes zoster. To the naked eye the only visible change was injection of the meninges. On section, abnormalities were found not only in the brain but in the cord as well. The latter showed a diffuse proliferation of the microglia and an infiltration of lymphocytes in the posterior horns. There was no perivascular demyelinization, but the nerve cells showed chromatolysis. In the anterior horns there was no inflammatory reaction, but the cells showed loss of Nissl's substance. The brain showed perivascular infiltration in the medulla, cerebellar nuclei, thalamus, and hypothalamus. The substantia nigra was not involved and there was no predilection for the periadqueductal grey

matter. There were numerous small perivascular hæmorrhages (more than in the spinal cord). The cytoplasm of the nerve cells showed hyaline changes and in the cortex a variable amount of chromatolysis was present, though perivascular hæmorrhages were absent. In Thalhimer's case the histological findings were similar and the main feature was infiltration with small round cells throughout the whole central nervous system.

These results show that the conception of herpes zoster as an infection of the nervous system as a whole, rather than as a localized disease of the posterior root ganglia rests on a firm pathological basis. It was put forward tentatively by Head and Campbell, who regarded herpes zoster as analogous to acute anterior poliomyelitis in having an initial febrile phase indicative of a general systemic infection, followed by signs of damage to a specific type of cell, and they suggested that it might be regarded as an acute posterior poliomyelitis. The evidence which has been accumulated since that time allows us to push this analogy further and to suggest that there may be a stage of generalization in which the virus is disseminated throughout the central nervous system. In the majority of cases the signs are only those of damage to the posterior root ganglion for which the virus has a special affinity. In a few cases, on the other hand, there is evidence that damage has been done to the rest of the nervous system, and it is this type of case which is considered in this paper. This view is also supported by the frequent occurrence of signs of meningitis in cases of herpes zoster, either in the form of headache, neck rigidity and a positive Kernig's sign, or a pleocytosis and an increase of protein in the C.S.F. The latter finding might possibly be more frequent than it is, if lumbar punctures were done in more cases of herpes zoster.

The first of the three cases described in the paper is an example of a lower motor neurone lesion due presumably to a spread of inflammation from the posterior root and the posterior horn to the anterior horn of the same segment. The distribution of muscular weakness (mainly lumber 2 and 3) corresponds well enough to the area of skin affected by the herpes. As has been seen already, such cases are not uncommon, though those affecting the lumbar segments seem to be the least common type. The cerebrospinal fluid findings were interesting, the slight xanthochromia and protein-cell dissociation being more suggestive of a neoplastic or vascular lesion than an inflammatory one. Such C.S.F. findings do, however, occur in the later stages of acute anterior poliomyelitis, and in this case the lumbar puncture was performed five weeks after the onset of symptoms. In other similar cases a moderate pleocytosis has been described (Carter and Dunlop, 1941). The progress of this case indicates that recovery does ultimately tend to occur, though it may be slow, intermittent, and incomplete, which agrees with the results in similar cases described by others. In this case the existence of some degree of facial weakness on the same side as the weakness of the leg and the herpes, and of symptoms such as insomnia, depression, and "muzziness" are at the least suggestive of a more widespread involvement of the nervous system.

The pathogenesis of these cases would seem to be that of an anterior poliomyelitis directly due to the herpes virus in which there is a considerable degree of recovery possible, probably greater than in acute infective anterior poliomyelitis, but in which some of the anterior horn cells nevertheless suffer irremediable damage. The available evidence suggests, however, that damage to the anterior root or even to the nerve trunk may also account for some of the cases of lower motor neurone paralysis following herpes zoster.

The second case represents an acute transverse lesion of the spinal cord with a paraplegia and loss of sphincter function occurring about seven to eight weeks after herpes zoster of the 10th and 11th dorsal segments. The mode and time of onset make it unlikely that in this case we are dealing with an inflammatory lesion due to spread of the herpes virus from the posterior root ganglion. It is much more likely that in this case there was some separate pathological process occurring either as a result of, or coincidentally to, the herpetic inflammation. In this connection the suggestion of Bruce (1907) that the wide extent of the lesion in many of the cases of spinal lesions following herpes zoster, might be accounted for by an inflammatory thrombosis of the postero-lateral or posterior cornual arteries is interesting. Furthermore, many of the lesions described in the brain stem and basal ganglia by Lhermitte and Vermes were hæmorrhagic and destructive in nature, though these authors emphasize that they were not associated with foci of degeneration and hyperæmia, and considered that they were not due to arterial or venous thrombosis. Finally, vascular dilatation and hæmorrhage are frequent findings in the spinal cord in cases of zoster. There would thus seem to be considerable support for the view that herpes zoster can lead to vascular lesions in the cord and brain, and it is possible to argue that in this case the acute myelitis might have been due to a thrombosis or hæmorrhage.

The fact, however, that there was a considerable degree of recovery followed by a further deterioration raises a further possibility. It is well known that herpes zoster can occur secondarily to some other disease of the central nervous system, such as trauma, neoplasm, or inflammation due to other agents, a condition known as "symptomatic herpes." In a case described by Kamman (1928) an intradural cyst was found on laminectomy, and Alajouanine and Griffith (1931) describe the case of a girl of 17 who developed typical signs of disseminated sclerosis two years after a "herpes myelitis." In our case the onset of the herpes and the paraplegia was preceded by a transient attack of blurring of vision; examination showed in addition to the paraplegia, nystagmus, doubtful

temporal pallor of the discs and intention tremor; in the C.S.F. there was a slight abnormality in the Lange curve with a negative W.R. These signs were not confirmed when he was seen two years later, but the possibility remains that his cord lesions might have been due to Disseminated Sclerosis, as in Alajouanine and Griffith's case. An acute transverse myelitis, while perhaps an uncommon feature of Disseminated Sclerosis, does occur (Wilson, 1940).

Though it is difficult to be certain in this case what the exact pathogenesis is, good evidence exists that zoster can cause lesions in the spinal cord affecting both the grey and the white matter, and which may be termed a zoster myelitis, though whether this condition is directly due to the action of the virus or to the indirect effect of vascular lesions must be left open.

The third case is perhaps the most interesting of the three. Following a herpes of the ophthalmic division of the trigeminal nerve, the patient developed a right hemiplegia with an aphasia three weeks after the appearance of the rash. The occurrence of an aphasia after herpes zoster has not, so far as we have been able to discover, been described before. It was of the expressive type and improved rapidly as did the hemiplegia. The C.S.F. showed a definite pleocytosis and increased protein, and in the examination carried out before admission, a xanthochromia. It is possible to conceive that the herpes zoster and the ensuing hemiplegia and aphasia were entirely unconnected and that their association was merely coincidence. It was unfortunately not possible to carry out complement fixation tests on the C.S.F. as was done by Schiff and Brain (1930), but nevertheless it seems difficult to account for the occurrence of a sudden hemiplegia in an otherwise healthy young adult of 26, with no evidence of arteriosclerosis elsewhere and no source of embolism. It therefore seems to us a reasonable assumption that the two conditions were associated and that the lesion was likely to be of the type described by Lhermitte and Vermes and by Faure-Beaulieu and Lhermitte (1929) in the brain stem and basal ganglia, rather than of that described by Biggart and Fisher (1938); that is, a localized hæmorrhagic or destructive focus affecting the left pyramidal tract in its course between the cortex and mid-brain. The symptoms and course are quite unlike those of the generalized encephalitis occurring in Biggart and Fisher's or Schiff and Brain's cases, where there was drowsiness, confusion, and disorientation rapidly progressing to coma and death; a type of clinical picture analogous to that seen in epidemic encephalitis, or the encephalitis due to the viruses of the acute exanthemata. The sudden onset, rapid recovery and the xanthochromia of the C.S.F. are in favour of a " vascular " type of lesion. In the cases described by Lhermitte and his colleagues, the picture was considerably complicated by the age of the patients (75 and 80), which suggest that senile arteriosclerotic changes might be responsible for the lesions, and by the presence of a positive W.R. in the C.S.F. of one of the cases. Although these authors make out a very good case for supposing that the lesions found post-mortem were in fact due to the herpes virus, though they might be predisposed to by the existence of vessels " which were weak, both easily ruptured and easily obliterated," their conclusions that such lesions always occur in old people, and that the state of the vessels is a necessary predisposing factor in their production, might seem to be invalidated by this case, where a lesion which appears to be of a similar type (though it has been impossible to verify this pathologically) occurs in a man of 26. The advanced age of the other cases of this type which have been described has made it difficult to assess prognosis fairly, but in young people it would not appear from this case to be uniformly bad, either as regards life or recovery of function. The site of the original zoster does not necessarily appear to have any connection with that of the brain lesion, since in both of Lhermitte's cases the herpes was spinal. However, in the cases of generalized encephalitis following zoster, Schiff and Brain point out that there may be meningeal symptoms with a local predominance corresponding to the site of the original zoster. In these cases they stress the possibility that the herpes may be " symptomatic " to an encephalitis of other ætiology, but conclude that cases in which the zoster clearly precedes the encephalitis can be considered as cases of zoster encephalitis. Biggart and Fisher consider that in such cases the virus may invade the brain along preformed nervous pathways, in view of the fact that the lesions in their case were particularly marked in the parietal cortex.

In all these cases of generalized encephalitis the lesions demonstrated at autopsy have in common the occurrence of perivascular hæmorrhages and destruction of the nerve cells. Several authors have commented on the fact that these features are characteristic of the lesions of herpes zoster wherever they occur, in the posterior root ganglia, spinal cord or brain. How far this can be taken to indicate that these lesions are vascular in origin is doubtful. While it seems clear that the herpes virus can give rise directly to inflammatory lesions in the course of a generalized invasion of the central nervous system, consideration of many of the cases described in the literature and perhaps especially cases 2 and 3 of this series, would seem to suggest that herpes zoster may lead to an inflammatory thrombosis or hæmorrhage, which is itself responsible for the sudden onset of a hemiplegia or paraplegia several weeks after the original infection. In support of this are the recent observations of Denny Brown, Adams and Fitzgerald (1944), who describe so complete a necrosis of both parenchymatous and interstitial tissues in the posterior root ganglia of a case of herpes zoster, that the appearance was that of an infarct necrosis. They conclude therefore that vascular occlusion was the cause, especially as they were able to demonstrate a thrombosis of the main vessel to the ganglion.

Summary

Three cases of herpes zoster complicated by paralysis and other symptoms indicative of involvement of the central nervous system are described and their pathogenesis discussed.

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BOOK REVIEWS

AN INTRODUCTION TO PHYSICAL METHODS OF TREATMENT IN PSYCHIATRY

William Sargant and Eliot Slater

(E. & S. Livingstone, Edinburgh, 1944. Pp. 171. 8*s.* 6*d.*)

In this short book, which is the first of its kind to appear, the authors have described the various physical treatments for psychiatric disorders which have been developed in the last 20 years. Much of the working principles and details that are provided, are not otherwise available in book form. The insulin treatment of schizophrenia, convulsion therapy, continuous narcosis, prefrontal leucotomy, and malarial therapy provide half the subject matter, and are described in a lucid manner, with plenty of practical guidance in technique which will prove invaluable to the student coming to psychiatry from a general medical training. The authors describe the indications for each treatment, the results that can be expected with suitable selection, the technique and the risks involved. They emphasize the importance of early insulin treatment for the schizophrenic and by quoting the relevant figures from the New York State hospitals may, we hope, discourage the continued use in these cases of convulsion therapy. The latter is the treatment of involutional depression in which condition a 70–90 per cent. chance of rapid recovery is offered. Physical treatment during the fit is favoured, and a form of straight-jacket for this purpose is described. It is emphasized that the uses of sleep therapy have been reduced by the introduction of specific treatments for schizophrenia and the depressive states, and the authors consider its chief use to be in manic states and in any very agitated patient as a preliminary to a specific therapy. It is most valuable in the acute anxiety states of war, but in general is to be regarded as a form of psychiatric first aid and nothing more.

The emphasis laid on diet and fluid intake for the narcosis patient and on the constitutional approach which led to the development of modified insulin treatment for the exhausted neurotic, recalls the Weir-Mitchell technique. Modified insulin therapy may well find a place in general medicine. The chapter on the uses of intravenous barbiturates adds nothing new to an already familiar technique, but the advice that " a cautious and sceptical attitude will save the doctor from swallowing all of his patient's hocum, hunting Snarks, and exploring nightmare's nests " is timely.

The discussion on diet, vitamins, and endocrines, although containing many practical therapeutic suggestions, is unfortunately diffuse and badly laid out. It is hoped that in a later edition this will be revised. It would appear preferable to deal with the administration of thyroid extract in this chapter rather than under " chemical stimulation." In giving a chapter the title " Treatment of cerebral dysrhythmia," the authors have boldly followed the Harvard school of electro-encephalography, though they admit that it is the fits or the behaviour disorder that are treated and that the dysrhythmia frequently remains unchanged while the patient improves. Although stimulating, as a guide to the treatment of epilepsy, this chapter could be greatly improved.

The authors support their book by an introduction, which is an excellent résumé of the theory of constitutional psychiatry, and by a final chapter which is perhaps an attempt to mollify the psychotherapists. But here psycho-analysis and all forms of " depth " therapy find little support. The authors conclude that " the somatic approach is only capable of improving the instrument that is in our hands; psychotherapy may have something to say of the aim towards which it is directed." Many will disagree with this and with other instances when a mechanistic behaviourism is assumed. However, in the main the authors have achieved what they attempted. This book is not academic. Even though it was written for the student and young clinician, some references and a bibliography would have been helpful. The style, always readable, is at times colloquial. Yet it is stimulating and is infused with an obvious therapeutic enthusiasm that is rare among the exponents of constitutional psychiatry. Undoubtedly it will be of great value to the post-graduate student. It is to be hoped that a second edition will follow in which greater attention is paid to those chapters which, admittedly, present the greatest difficulties.

A TEXT BOOK OF PSYCHIATRY FOR STUDENTS AND PRACTITIONERS

D. K. Henderson and R. D. Gillespie

(Humphrey Milford, Oxford University Press. Sixth Edition, 1944. Pp. 719. 25*s.*)

This book is probably the best known work on psychiatry by British authors, and represents for the most part the standard teaching in this country. So much regarding the exact mechanism of mental disorder is unknown, that it is a pleasure to read a book on the subject which is completely free from dogma and over-simplification. At the same time the authors have succeeded in presenting the subject in a lucid manner with a minimal use of less familiar phraseology. The book is thus especially suitable for those wishing to acquire sound fundamentals in this branch of medicine.

Important new features of this edition include discussions on pre-frontal leucotomy and electric shock therapy. That bilateral division of the fronto-thalamic fibres in selected cases of mental disorder is frequently followed by extremely beneficial results is no longer to be doubted, but it is clear that the operation has limited indications, and in a few cases unfortunate results. The present view is expressed that the pure obsessional states are likely to respond the most favourably, whilst those states characterized by hallucinations are less benefited; but that neither is a real indication for surgery unless every other method of treatment has failed, and considerable occupational disability has resulted.

The text has been re-arranged to some extent, a noticeable alteration being the inclusion of the so-called paranoid forms of schizophrenia with paranoic and paranoid reaction-types instead of with the schizophrenic reaction-types as hitherto. Many will share the authors' view that this conception has advantages, particularly with regard to prognosis.

The chapter on psychosis and psycho-neurosis in war is detailed enough, and will prove useful to the serving doctor.

NEUROLOGY OF THE EYE, EAR, NOSE AND THROAT

E. A. Spiegel and I. Sommer

(Grune and Stratton, New York. 1944. Pp. 667)

There is need for a book or books which present the facts, so far as they are known, concerning the borderland between neurology, otolaryngology and ophthalmology. The authors are to be congratulated on a well-documented book which goes far to fill this need. It is indeed helpful to those seeking knowledge that the authors have not only provided a very full list of

references at the end of the book, but have also indicated at the end of each chapter the more important works on the subject dealt with. There is little doubt that this book will be welcomed by many and accepted as a textbook, worthy of study, in the fields of neurology, otology and ophthalmology.

It is published in clear type. The reproductions are on the whole good and the X-ray pictures are of sufficient clarity for the abnormality to be readily seen. The list of references, 1719 in all, is comprehensive and the index adequate. This is a book worthy of commendation.

RECENT ADVANCES IN NEUROLOGY AND NEURO-PSYCHIATRY

W. Russell Brain and E. B. Strauss

(J. and J. Churchill, London, 1945. Pp. 363. 18*s*.)

In a somewhat extensive preface the authors give a dissertation on their reasons for adjusting the title of this book to that of " Recent Advances in Neurology and Neuro-Psychiatry". It is indeed difficult to understand or follow their argument for the inclusion of " neuropsychiatry". Their reference to the part played by the autonomic system in hormone regulation and their following statement of the consequent necessity that neurology and psychiatry must overlap are surely open, if not to criticism, to considerable misunderstanding. And if they state " the whole of the book is neuropsychiatric " then why include neurology in the title? As the book contains much about neuro-physiology, neuro-surgery and neuro-pathology, should these subdivisions of neurology be left out of the title? The new title is indeed unfortunate.

There is no doubt that the authors are to be congratulated in presenting in a readable and concise form the recently acquired knowledge as is to be found in journals published throughout the world. Their assessment of the importance of the various contributions is, however, on occasion open to question. Is it indeed a fact that prefrontal leucotomy may be looked upon as a cure for " biogenetic psychoses and psycho-neurotic reaction formations "? A more critical approach to this subject is surely still necessary. Even the authors themselves in their summary of this chapter on prefrontal leucotomy state that this form of therapy is justifiable when other forms of treatment have failed and when many other aspects have been weighed up. This does not appear to substantiate their statement in the introduction to this chapter. In the chapter on electrical convulsant therapy does the inclusion of a photograph of the apparatus add useful information? It cannot be said that the photograph is helpful either to the clinician in assessing the value of the therapy or to the expert in obtaining the lay-out of the circuits.

In the chapter on disorders of muscle there is an excellent description of the muscular disorders associated with thyroid disease and a fair survey has been given of the value of thymectomy in myasthenia gravis. The following chapter deals with sciatica and the authors take the view that this condition is caused by one of a number of pathological conditions. There will be many neurologists who will hesitate to accept the term sciatica as covering many of the conditions listed. The authors draw attention to the advisable need for caution in assessing the results of operation in the treatment of patients with a herniated intravertebral disc.

This is a useful book to act as an introduction and guide to the recent additions to literature in the field of neurology. It should not, however, replace or deter the study of the original papers.

NEURO-OPHTHALMOLOGY

Donald J. Lyle

(Chas. C. Thomas, Springfield, Ill. 1945. Pp. 395. $10.50)

In the preface the author may be interpreted as indicating that his book is written mainly for the purpose of helping the ophthalmologist when requested to consult with neurological colleagues. The danger, therefore, to which the author may submit himself when he attempts to cover the two specialities—ophthalmology and neurology—is that neither are dealt with in a manner satisfying to the critically interested medical man in either.

In nineteen chapters a very wide field is covered from the embryological development of the neural elements of the eye through a somewhat short survey of the physiology of vision to several chapters concerned with diseases of the nervous system. The reviewer finds himself in difficulties in assessing the merits of a book so arranged. He regrets that a more adequate presentation of the anatomy and physiology of vision, especially that of the peripheral fields, has not been included. It is indeed difficult either by reading the book or referring to the index to find out the character of the field defects which may be met with in small focal lesions in the region of the calcarine fissure. Profusely illustrated, especially with stereophotographs of the fundus, with clinical photographs and with photographs of brain dissections and pathological specimens, these photographs do not replace a well-written description of many conditions. Is a picture of an anencephalic child helpful or needful? Does a picture of a child in the last stages of amaurotic family idiocy show anything but a wasted child? Do X-ray reproductions which reveal little or no detail add value to the book? These are questions which the reviewer hesitates to answer in the affirmative.

There is, however, collected in this book information which should be readily available to ophthalmologists and accordingly this book may find approval. It is, however, uncertain whether neurologists will agree that the book fills a want. Possibly, in a second edition, the author may see his way to reduce the number of photographs, to lengthen the text and to improve the index.

DISEASES OF THE NERVOUS SYSTEM

F. M. R. Walshe

(E. and S. Livingstone, Edinburgh. Fourth Edition, 1945. Pp. 360. 15*s*.)

This is a fourth edition, an indication of the popularity of this textbook on diseases of the nervous system. The author in the past has taken advantage of each new edition to revise and amplify and again he has done so. This edition has benefited by the alterations and additions made to the text.

In view of the interest displayed by physicians and surgeons in the part possibly played by disturbances of the autonomic nervous system in various disease conditions, the absence of a short statement as to the function of and signs arising from disturbance of the autonomic nervous system is disappointing. The autonomic nervous system is referred to neither in the chapter on anatomical or localizing factors, nor in that on examination of the nervous system.

It remains a textbook worthy of recommendation to student and practitioner.

THE EXAMINATION OF REFLEXES

Robert Wartenberg

(The Year Book Publishers, Chicago, 1945. Pp. 222. $2.50.)

A student may well be perplexed to find a book of 182 pages devoted entirely to the examination of reflexes. The merit of this book is not so much in the description of the many reflexes mentioned as in the author's insistence that many supposedly different reflexes are in fact not infrequently one and the same reflex. He has helped to clear away much unnecessary superstitions which sometimes clouded the proper interpretation of abnormalities of reflex activity. For this reason the student of neurology might well benefit from reading this book.